

Anomalous Self-Experience in Schizophrenia

Associations with diagnosis, suicidality and neurocognition

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Oslo 2012

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*Series of dissertations submitted to the
Faculty of Medicine, University of Oslo
No. 1368*

ISBN 978-82-8264-360-3

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Printed in Norway: AIT Oslo AS.

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Acknowledgements

The present study was established in 2007 by the Network for Early Stage Psychosis Research (Helse SørØst Kjernekompetansemiljø), under the management of Professor Ingrid Melle. She inspired me to design an independent PhD project within a larger project on psychoses, the TOP (Thematic Organized Psychosis) research group (Forskningsgruppe Helse SørØst), headed by Professor Ole Andreassen. They wanted to initiate more research activity among the clinicians outside the university hospitals.

During almost 20 years as a clinician, mainly with patients suffering from psychoses, I heard a lot of stories about how they experienced their psychoses. So when I got the opportunity to do some research, I chose to study the subjective experiences of patients suffering from schizophrenia.

I knew that Dr. Med. Paul Møller, working in the research milieu on self-disturbances at the Unit of Mental Health Research and Development, Division of Mental Health and Addiction, Vestre Viken Hospital Trust, had done a study on the phenomenology of the prodrome in schizophrenia, and that he had a special interest in subjective experiences in schizophrenia. Therefore I got in contact with him and asked him to be my supervisor.

The study was funded by the research unit at Innlandet Hospital Trust (Sykehuset Innlandet HF (SIHF)) and the South-East Health Authority, and took place in the Department of psychosis and rehabilitation, Division of Mental Health at SIHF.

The project has been dependent on many skilled contributors. It was carried out in close cooperation with another research project on psychotic disorders within SIHF, by Unni Bratlien, M.D. We established a common research group including Professor Ingrid Melle, M.D., Professor Ole Andreassen, M.D., Dr. Med Paul Møller, M.D, Dr. Med. Lars Lien, M.D. and Dr. Psychol Merete Øie, neuropsychologist.

My supervisors have been Dr. Med. Paul Møller, M.D., Professor Ingrid Melle, M.D. and Dr. Med Lars Lien, M.D.

First of all I want to thank my main supervisor Dr. Med Paul Møller who introduced me to this challenging research field and very generously encouraged and supported me all through the study. He always had time to answer my questions and contributed with major insight into this field, which was of vital importance to carry it through.

I am also indebted to my co-supervisor Professor Ingrid Melle for including me in the Network for Early Stage Psychosis Research and giving me particularly important and timely support to my project idea at an early stage. She gave me constructive supervision throughout the whole project. She always kept an experienced “bird’s-eye view” on this study.

I also want to thank Dr. Med. Lars Lien, who has been my project leader and the research group’s important connection to SIHF. He helped me designing the project and contributed to several manuscript revisions.

At an early stage of this project Professor Ole Andreassen, MD included me in the TOP research group and helped me designing the study. He also gave me important feedback on manuscript revisions.

Dr. Psychol Merete Øie supervised the test assistants, who did the neurocognitive testing. She also gave a very important contribution concerning the neurocognitive aspect of this project, especially as a co-author on my third paper. She was very efficient and always there when I needed her advice and feedback.

At a workshop in Heidelberg in 2010 I met Andrea Raballo M.D., who at that time was working at the Danish National Research Foundation: Center for Subjectivity Research and the Department of Psychiatry, Psychiatric Center Hvidovre, University of Copenhagen, Denmark.

During the final part of the project Andrea Raballo, MD helped me with statistical issues and gave important comments during several manuscript revisions.

I also want to thank Unni Bratlien, M.D. for her important cooperation during establishing the project in SIHF and during inclusion of the patients.

The study would not have been possible to conduct without the psychologists, Kristine Lund, Erik Winter and Gunhild Winter who did the neurocognitive testing.

Thanks to Josef Parnas for all the inspiration I got, attending his EASE workshops and reading his papers.

My two sons, Sindre and Erlend, also deserve thanks for being so patient and letting me work in the evenings and weekends. Their love, laughter and humour also sometimes helped me forget the project and gave me important breaks.

Thanks also to my boyfriend, Lars, for his love and comfort during this period.

Finally, I am most grateful to all the patients for contributing so generously with their lived life experiences and for their endurance during all the clinical, neurocognitive and somatic assessments.

Summary

Increasing and robust empirical evidence indicates that certain anomalous subjective experiences in the form of non-psychotic disturbances of the basic sense of self (i.e. self-disorders, SDs) might be specific vulnerability markers for schizophrenia spectrum disorders (Møller and Husby, 2000; Parnas et al., 1998; Raballo, 2009; Raballo et al., 2011; Parnas et al., 2003). The term self-disorders (SDs) indicates that these phenomena are basic disturbances of the person's subjective experience of his own identity or "self" (Sass and Parnas, 2003).

The main purpose of this study is to contribute to more knowledge about the phenomenon of SDs in the early stages of psychosis. We wanted to study the more basic phenomena of psychotic psychopathology through assessing occurrence, level and type of SDs close to the onset of psychosis, and subsequently linking the disorders to differences in clinical presentation.

Early diagnostics and treatment is of importance in psychotic disorders.

Current diagnostic manuals (e.g. DSM-IV and ICD-10) do not differentiate clearly between different psychotic disorders in the early stages of the illnesses due to overlap in clinical symptoms and behavioural manifestations. Thus, one purpose of this study is to contribute to better diagnostic precision in these early stages of psychosis through more knowledge about the phenomenon of SDs.

Additionally, we know that one of the major complications associated with schizophrenia is suicidal behaviour, and the risk factors identified until now cannot explain why the suicide risk is particularly high in the early phases of the disorder. Suicidality is multidetermined and our hypothesis is that suicidality may be partly motivated by SDs.

Finally, both SDs and neurocognitive deficits have been suggested to be core features of schizophrenia, so we also wanted to study tentative relationships between them.

We therefore posed the following questions:

Can SDs discriminate between schizophrenia spectrum disorders (schizophrenia, schizophreniform disorder and schizoaffective disorder), bipolar disorders (bipolar disorder I and NOS) (BD) and other psychotic disorders usually classified outside of the (narrow) schizophrenia spectrum (delusional disorder, brief psychotic disorder and psychosis NOS) (OP) in the early phase of the treated course of psychotic disorders?

Is current suicidality related to SDs in first treatment schizophrenia patients, and how is the relationship between suicidality, depression and SDs in these patients?

Is there any relationship between neurocognitive dysfunctions and SDs in the early phase of schizophrenia?

The study involved all treatment facilities in two neighbouring Norwegian counties. Inclusion criteria was being between 18 to 65 years, and being consecutive in- or outpatient referred to first adequate treatment (FAT) for psychosis. During 2008 and 2009 a total of 91 patients early in their treatment course completed the full protocol including clinical assessments and neurocognitive assessments. SDs were assessed according to the EASE (Examination of Anomalous Self-Experience) manual (Parnas et al., 2005b).

Because EASE is a relatively new instrument, we also wanted to test if it is a reliable and internally consistent clinical tool for the assessment of anomalous subjective experience in patients referred to (FAT) for psychosis.

We believe that the additional perspective of SDs has large potentials to improve diagnostic validity in the early course of illness, and thus contribute to an earlier and more targeted treatment of psychotic disorders.

Suicide prevention in patients with first episode schizophrenia is important, and these results can shed some light in the search for risk factors for suicide in this patient group.

More knowledge about the relationship between neurocognitive deficits and the person's experience of his/hers thinking processes might improve the communication with the patients about their neurocognitive deficits and aid treatment.

List of papers

1. Examination of anomalous Self-Experience in First-Episode Psychosis: Inter-Rater Reliability
2. Selective aggregation of self-disorders in first treatment DSM-IV schizophrenia spectrum disorders
3. The association between anomalous self-experience and suicidality in first episode schizophrenia seems mediated by depression
4. The association between Self-Disorders and Neurocognitive Dysfunction in Schizophrenia.

Abbreviations

BD	Bipolar disorders (Bipolar I and Bipolar NOS)
BSs	Basic Symptoms
BSABS	Bonn Scale for the Assessment of Basic Symptoms
CAARMS	Comprehensive Assessment of at Risk Mental States
CDSS	Calgary Depression Scale for Schizophrenia
CVLT	California Verbal Learning Test
D-KEFS	Delis-Kaplan Executive Function System
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders Fourth Edition
DUP	Duration of Untreated Psychosis
EASE	Examination of Anomalous Self-Experience
FAT	First Adequate Treatment
FCQ	Frankfurt Complaint Questionnaire
GAF	Global Assessment of Functioning Scale-Split version
GAF-F	Global Assessment of Functioning Scale-Functioning-subscale
GAF-S	Global Assessment of Functioning Scale-Symptom subscale
IQ	Intelligence quotient
NART	National Adult Reading Test
OP	Other psychoses (delusional disorder, brief psychotic disorder and psychosis NOS)
PANSS	Positive and Negative Syndrome Scale for Schizophrenia
SDs	Self-Disorders
SCID-I	Structured Clinical Interview for DSM-IV Axis I disorders
SIHF	Sykehuset Innlandet Helseforetak (Innlandet Hospital Trust)
SIPS	Structured Interview of Prodromal Syndromes

SCI-PANSS Structured Clinical Interview for the PANSS

SPI-A Schizophrenia proneness instrument for adults

SZ Schizophrenia spectrum psychoses (schizophrenia, schizophreniform disorder and schizoaffective disorder)

TOP Thematically Organized Psychosis Research Study

UHR Ultra-High Risk

WAIS-III Wechsler Adult Intelligence Scale-III

WASI Wechsler Abbreviated Scale of Intelligence

WMS-III Wechsler Memory Scale-III

YMRS Young Mania Rating Scale

1. INTRODUCTION

Psychoses

Mental disorders are now the largest causes of disability in the developed countries, and the psychotic disorders are ranked among the leading contributors to the total burden of disease worldwide. They are devastating disorders, often emerging during early adulthood and lasting for the entire adult lifespan. The consequences are often reduced capacity for productivity, creativity and relatedness. They also represent major challenges to society due to significant treatment costs.

During the last two decades we have seen an emerging interest in prevention through early detection and intervention (McGorry et al., 2010;McGlashan and Johannessen, 1996). Studies have shown that patients with shorter duration of untreated psychosis have less suicidality, lower levels of negative symptoms and better social functioning (Melle et al., 2006;Larsen et al., 2010;Melle et al., 2008;Marshall et al., 2005). This increasing focus on early detection of psychotic disorders has boosted a parallel need for developing suitable clinical tools that can maximize risk stratification and guide differential diagnosis in the early phases, both prepsychotic and psychotic (Raballo and Laroi, 2009;McGorry et al., 2007;Parnas, 2005).

Historically, the term “psychosis” dates from 1845(Beer, 1996) and has received a number of different definitions. In 1899 Kraepelin divided this concept into dementia praecox and manic-depressive psychosis (Angst, 2002). Today, more than 100 years later, this distinction between schizophrenia (dementia praecox) and bipolar disorder (manic depressive psychosis) remains almost the same. However, this distinction has been questioned, and during the last years there has been a discussion whether this is categorically different disorders or if they exist on a continuum. One argument for the continuum theory is that they show overlapping symptoms.

The available diagnostic systems (e.g. DSM-IV and ICD-10), however, are not a product of conceptual analyses and empirical evidences, but defined through consensus with the purpose of improving reliability. The diagnostic criteria are based on clinical symptoms and their behavioural manifestations, and have not been established through basic analyses of psychopathology or knowledge of biological signs linked to the underlying aetiology of the disorders (Jansson and Parnas, 2007) . Due to the indistinct, unspecific and overlapping nature of conventional clinical symptoms, the available diagnostic systems do not differentiate well between schizophrenia and, psychotic bipolar disorder, nor do they offer help in capturing differentiating symptoms of incipient as well as established psychosis (Meyer et al., 2005). Existing prodromal or ultra-high risk (UHR) criteria also do not allow for a clear prepsychotic differentiation between various forms of psychosis (Cannon et al., 2007; Correll et al., 2007; McGorry et al., 2003). While the Schizophrenia proneness instrument for adults (SPI-A) (Schultze-Lutter et al., 2007a) was conceived to particularly identify patients at risk for developing schizophrenia, other instruments like Structured Interview of Prodromal Syndromes (SIPS) and Comprehensive Assessment of at Risk Mental States (CAARMS), are less specific and identify early clinical features predictive of several forms of psychotic disorder including affective psychoses (Cannon et al., 2007; McGorry et al., 2003; Yung et al., 2005).

The focus of the present thesis will be schizophrenia spectrum disorders (i.e. schizophrenia, schizophreniform disorder and schizoaffective disorder) (SZ), bipolar disorders (i.e bipolar I and bipolar NOS) (BD) and other psychoses (i.e. delusional disorder, brief psychotic disorder or psychotic disorder NOS) (OP).

Diagnoses according to DSM-IV

Schizophrenia spectrum disorders

Schizophrenia is a group of mental disorders with symptoms which are commonly divided into positive, negative and disorganized symptoms. Positive symptoms are based on the occurrence of productive signs of disturbance and include delusions and hallucinations. Negative symptoms are deficits like affective flattening, alogia and avolition, whereas disorganized symptoms are disorganized speech and behaviour.

The DSM-IV and ICD-10 classifications of diagnoses are based on description of the occurrence of such symptoms. According to DSM-IV, continuous signs of the disorder must persist for at least 6 months, the patient has to show functional decline and the disturbance must not be due to effects of a substance or a medical condition.

Schizoaffective disorder is a disorder that meets the criteria for schizophrenia and at least one mood episode, in specified constellations. Affective symptoms must be present for a substantial duration of the illness.

Schizophreniform disorder is, according to DSM-IV, a disorder that also meets the criteria for schizophrenia, but the duration of the psychotic symptoms is shorter and functional decline is not required.

Bipolar disorders

Bipolar disorder is characterised by the presence of discrete periods of abnormal mood and activation that define depressive, manic or hypomanic episodes. The DSM-IV definition relies on the identification of individual mood episodes occurring over time. In the current study we include patients with at least one manic or mixed episode.

Mania is an episode lasting at least one week, (or less if hospitalization is required). During this period three or more of the following symptoms are present: inflated self esteem or grandiosity, decreased need for sleep, talkative, flight of ideas or subjective experience that thoughts are racing, distractibility, increase in goal-directed activity or psychomotor agitation, and excessive involvement in pleasurable activities with high potential for painful consequences. According to DSM-IV, the disturbance leads to impaired function, psychotic symptoms or hospitalization, and must not be due to effects of a substance or a medical condition.

A mixed episode is defined as a period of at least one week, in which the criteria are met both for a manic and for a major depressive episode.

Other psychoses

Delusional disorder is, according to DSM-IV, characterised by one or more nonbizarre delusions that persist for at least one month. If hallucinations are present, they have to be associated with the delusions. Behaviour is neither bizarre nor odd, and there is no marked functional decline except for dysfunction from the direct impact of the delusions. The delusions are not due to the effect of a substance or a general medical condition.

Brief psychotic disorder involves the sudden onset of psychotic symptoms. It lasts less than a month, and the individual show full recovery.

Psychotic disorder NOS is a category of disorders with psychotic symptomatology that does not meet the criteria for any specific psychotic disorder.

Self-disorders (SDs)

General description and history

It has been asserted from several perspectives for a long time that schizophrenia involves profound transformations of the self. Issues of this altered self-experience has been described in literature from sources ranging from existential psychiatry, psychoanalysis, phenomenology, psychosocial rehabilitation, and dialogical psychology (Lysaker and Lysaker, 2010).

Converging empirical evidence also indicates that certain anomalous subjective experiences in the form of non-psychotic disturbances of the basic sense of self (i.e. self-disorders, SDs) might be specific vulnerability markers for schizophrenia spectrum disorders (Møller and Husby, 2000;Parnas et al., 1998;Parnas et al., 2003;Raballo et al., 2011). Despite this, SDs has not been mentioned in the diagnostic criteria for schizophrenia of neither DSM-IV nor ICD-10.

Phenomenologically, we can describe the sense of self (one might say identity feeling), on three hierarchical, but intertwined levels: the pre-reflexive, reflexive and narrative self (Parnas and Handest, 2003). The most basic level of the self is the implicit, silent, pre-reflective egocentricity, which is inseparable from and built-in in the subjective experience itself. The reflexive self is the explicit awareness of an “I” that is largely stable over time. The narrative self is the experience of the self as having special characteristics, personality and narratives, and only at this level we can talk about “self-image” and “self-esteem”. The term anomalous self-experience or self-disorders (SDs) in this study refers to disturbances at the most basic level, the pre-reflexive self. This is fundamental disturbances of first-person perspective, which means deficiency in the sense of being a coherent subject, a self-coinciding centre of action, thought and experience (Sass and Parnas, 2003). SDs are subtle, mainly trait-like disturbances of a person’s experience of him- or herself as a vital subject naturally immersed in the world and remaining one and the same through time. These disturbances are seen as distortions taking place in the fundamental levels of

consciousness, afflicting the very way experiences are structured (as mine). Later, these disturbances can (but not necessarily), through conscious personal attributions, develop into manifest delusions or hallucinations. Initially it is the structure (ipseity, subjectivity) of the experience that is changed, not yet the thematic content. The change in content, like psychotic or bizarre delusions, can be seen as a (possible) consequence of the change in form.

Even though bizarre delusions play a major role in the contemporary diagnostic systems (i.e. DSM-IV and ICD-10) and this phenomenon is well described and acknowledged as a core feature of schizophrenia, it is still not consensually known *how* such bizarre ideas can arise and why they emerge so often in some psychotic conditions and not in others.

One example is delusions of influence. Using the frame-work of anomalous self-experience, this can be characterised as a weakening, loss or distortion of subjectivity. The person loses the sense of basic identity, of having an inner core or of being “oneself”, and as a consequence loses the sense of ownership of his mental events (thoughts, feelings, perceptions, bodily experiences and actions). Over time this can give the person increasing convictions of being manipulated and controlled by external forces, and finally it can develop into delusions of control or influence.

SDs as nonpsychotic anomalies of schizophrenia were already described at the turn of last century, both in classic literature and in phenomenological psychiatry (Parnas and Handest, 2003).

Prominent scientists and clinicians like Kraepelin, Bleuler and Schneider recognized that the basic human identity and consciousness are disrupted in schizophrenia.

French psychiatrists published case histories characterized by profoundly altered self-experience of patients that today would be diagnosed as suffering from schizophrenia.

Bleuler considered “basic disorder” of personality as a core feature of schizophrenia (Bleuler, 1911), while Kraepelin declared that a disunity of consciousness, “an orchestra without a conductor”, was the fundamental feature of schizophrenia (Kraepelin, 1896;Kraepelin, 1913).

Berze suggested that subtle alteration of self-consciousness was the primary disorder in schizophrenia, and that it was most easily detectable in the incipient cases (Berze, 1914).

Jaspers made a list of experiential modes in which a self is aware of itself (self-activity, -unity, -identity and -demarcation) (Jaspers, 1923).

Scharfetter modified Jaspers list to include, in order of increasing complexity: vitality, activity, continuity, demarcation and identity (Scharfetter, 1980).

Schneider mentioned a “loss of egoboundaries” in his description of passivity phenomena (Schneider, 1959).

Somewhat later, an important contribution to this field came from Huber, Klosterkötter and their colleagues in Germany. In prospective and retrospective studies they investigated and described subtle (non-psychotic) affective, cognitive, perceptual, motor and bodily disturbances, which they named Basic Symptoms (BSs). Many of these symptoms are considered specific to schizophrenia (Klosterkötter et al., 1997;Gross and Huber, 1986;Huber and Gross, 1989).

The BSs are compiled and described in the Bonn Scale for the Assessment of Basic Symptoms (BSABS) (Gross et al., 1987). BSABS is a list of symptoms, including some SDs, but it also includes other accompanying manifestations of schizophrenia, such as affective-dynamic disorders (e.g. reduced stress tolerance to daily tasks, increased impressionability) and neurovegetative symptoms (e.g. sleep disorders).

In Norway, Møller published a qualitative thesis, containing in-depth interviews with 19 first-onset schizophrenia patients and their relatives. He found two tentative core dimensions: “disturbance of perception of self” (=disturbance of ipseity) and “extreme preoccupation by and

withdrawal to overvalued ideas” (=hyperreflexivity) (Møller and Husby, 2000). At about the same time, but independent of the Norwegian study, a pilot study in Copenhagen of 19 first-onset patients with schizophrenia demonstrated almost identical prodromal profiles (Parnas et al., 1998). A study from 2003 compared lifetime prevalence of the BSABS-defined anomalies of subjective experience between patients with residual schizophrenia and psychotic bipolar illness in remission. They found that the disorders of self-experience were the most significant discriminators between the two diagnostic groups (Parnas et al., 2003).

The EASE

Until recently, there was no specific instrument that allowed a comprehensive, guided clinical mapping of SDs. However, a few years ago with the publication of a dedicated tool, the Examination of Anomalous Self-Experience (EASE) (Parnas et al., 2005b), we got such an instrument specifically focussed on the assessment of SDs. The development of EASE was partly motivated by the clinical work at the University Department of Psychiatry of Hvidovre Hospital in Copenhagen, but particularly by the two independent clinical studies mentioned in the previous section (Parnas et al., 1998; Møller and Husby, 2000). Both these studies showed that – already in the prodromal phase - schizophrenia spectrum disorder patients report a wide range of disturbing, not-yet psychotic changes in the very experience of self and identity. The authors of the EASE were informed by classic psychopathological descriptions of these phenomena, and they were inspired by the work of the German research group of Huber, Gross, Klosterkötter, Schultze-Lutter, and their colleagues (Klosterkötter et al., 1997; Gross et al., 1987). EASE is a symptom checklist that comprises five domains: 1. Cognition and stream of consciousness. 2. Self awareness and presence. 3. Bodily experience. 4. Transitivity and

demarcation. 5. Existential reorientation. This represents a wide variety of anomalous self-experiences condensed into 57 main items and several sub-items.

There are some partial overlaps between the EASE and the BSABS (28 main items) which are listed in the manual, especially in domain 1 (Cognition and stream of consciousness) and some other single items, like cenesthetic experiences (unusual bodily sensations). Some EASE items are also similar to items in the (SIPS) (McGlashan et al., 2001), e.g. thought interference, perceptualization of inner speech or thoughts and cenesthetic experiences. Notably, the EASE assesses the most basic, pre-reflective level of self, i.e. a level of self-experience which escapes more extended and articulated levels of self-description, such as those captured by the Scale to Assess Narrative Development (NART) (Lysaker et al., 2009) and the Metacognition Assessment Scale (MAS) (Semerari et al., 2003).

Domain 1

Cognition and Stream of Consciousness

The focus in this domain is on the sense of consciousness as continuously and silently flowing over time, belonging to one subject and directly given in an abstract way. Normally, the thoughts should not be experienced as having physical qualities like direction, space, movement or location. So this is more about *how* the person is thinking, not so much *what* he is thinking. For example, some patients in this study experienced that they had thoughts in their head that seemed unfamiliar. “I know these thoughts are mine because they take place in my head, but I do not recognise them as my own thoughts”. Some experienced that the thoughts also had acoustic or auditory qualities, like hearing their own voice in their head while thinking. If a person has thoughts in his head that he does not recognise as his own thoughts, and these thoughts have auditory qualities, it is obvious that some can experience this as if they are audible thoughts coming from another source (in further elaborations possibly attributed as thoughts put into his

head, or voices coming from another source). A lot of patients, actually had difficulties in distinguishing between auditory hallucinations and their own thoughts. There were also some who felt as though others might be able to hear their thoughts.

Domain 2

Self-Awareness and Presence

This domain describes experiential anomalies related to changes in the normally unreflected self-presence, embeddedness in the world, and first-person perspective. A lot of the patients in this study were uncertain about their own existence. “Nobody takes notice of me; it is like being invisible, so perhaps I am dead?” If someone is unsure about this, he can of course also be unsure about everything else he is experiencing. This leads to huge existential questions like: “If I am not here, who is experiencing this, and who is thinking these thoughts? Is it possible that the world is real, but I do not exist? Or maybe I exist, but the rest of the world, including other people, does not?” This is sometimes accompanied by an extreme feeling of loneliness. “I must convince myself that I am still alive and that the world is real, if not, I am completely lost and alone”. Some feel that they exist, but they are not themselves anymore. “When I hear the music of Elvis, it *feels like* I am Elvis. Sometimes it feels so real that I actually think I am Elvis”. Distorted first person perspective can also be experienced as being an observer to ones own life. “It is like looking through a camera lens where I see my life as a movie.”

Domain 3

Bodily Experience

This domain focuses on aberrations in the normal experience of psychophysical (body-mind) unity and a normal feeling that the body is both a physical object and an abstract subject at the same time. Some patients in this study felt as if the body was merely an object that changed in strange ways. “Suddenly I feel as if my body has become very big or very small.”

Some did not recognise their own mirror-image. “I look in the mirror and I see someone who looks like me, but in a way, it is not me, because the mirror image has a different personality. When I stand before the mirror, I move my hand to see if the mirror-image also moves. If that is the case, I am sure that the mirror-image is mine.”

They could also feel that their bodies melted or fell apart. “A nurse at the ward said that I had a breakdown. I became very scared, because I suspected that my body had fallen apart.” Some experienced de-automatization of movement. “I have to focus on every single movement of the body. Therefore I am now, much too slow to play soccer, even though I used to be a good soccer player.”

Domain 4

Transitivity and Demarcation

This domain focuses on loss or permeability of self-world boundary. These experiences are closely related to self-consciousness and presence, but are listed here because of their quite articulated symptomatic nature. Some patients in the current study experienced confusion with the other. “I am provoked by the fact that other people do the same thing as me, because I begin to think that they can be me“. Others felt threatened by bodily contact, and some also had an overwhelming feeling that their whole existence was threatened.

Domain 5

Existential reorientation

This refers to a fundamental change in the patient’s metaphysical worldview and hierarchy of values. In this domain, the experienced self-awareness is existentially and behaviourally expressed. Solipsistic experience like self-centrality belongs to this domain. “I have the strange feeling that everything that happens is built up just for me, like a scene, because someone wants to test me. It feels like Japan does not exist, because I have not been there, but if I go there and

see it with my own eyes, I will be convinced that it exist.” Some explain this feeling as if they are in a “reality show” where everybody else are actors and the surroundings are stage sets “like the in the movie Truman show”.

The experiences described above are by definition not psychotic. In the early course of the illness, the patients define them generally as strange and unfamiliar (“as-if” experiences). Most of them try to resist the feeling that the experience could be real. Often they try to behave as usual even though everything is very strange. However, some say they are becoming more and more convinced that their “private reality” is real, and this might be the next move towards delusional convictions.

Suicidality in schizophrenia

Suicidal behaviour and subsequent high risk of suicide are major complications in schizophrenia. The lifetime risk of suicide in patients diagnosed with the disorder is about 5% (Palmer et al., 2005), while 20-30% attempt suicide (Fenton et al., 1997;Radomsky et al., 1999) The risk of suicide is highest during the early phases of the disorder (Palmer et al., 2005;Nordentoft et al., 2002). Up to 25% of first-contact patients have made one or more previous suicide attempts (Barrett et al., 2010b). Untreated patients appear to have a particular high risk for violent attempts, emphasizing the importance of early treatment (Barrett et al., 2010b;Melle et al., 2006;Melle et al., 2010). Several predictors of suicidal behaviour have been identified. These include risk factors also seen in the general population, such as being male, abusing substances, living alone, being unemployed, being depressed, experiencing hopelessness and/or having a history of previous suicidal ideation/attempts (Breier and Astrachan, 1984;Burgess et al., 2000;Hawton et al., 2005). Several of these risk factors are more prevalent in patients with

schizophrenia compared to the general population. Also risk factors more specific to patients with psychotic disorders have been identified, including longer duration of untreated illness, more severe illness course, non-adherence to treatment, and better insight (Hawton et al., 2005;Barrett et al., 2010a). The level of positive psychotic symptoms is mostly found to be unrelated to suicidal behaviour (Hawton et al., 2005). Findings are however not consistent, and risk factors have a low predictive power, making it difficult to initiate targeted suicide prevention in clinical settings.

Risk factors identified until now cannot explain the high suicide risk in the early stages of the disorder. One possible exception to this is depression, which is more prevalent at this point of time (Romm et al., 2010), but then again, why are patients more depressed in the early stages? Recent studies have thus focused on patients' subjective experiences in general (not self-disorders), finding that low satisfaction with life, hopelessness, negative self-appraisals, loneliness, preserved insight and negative views and stigma connected to severe mental disorders are associated with suicidal behaviour in these early stages (Melle et al., 2010;Barrett et al., 2010a;Skodlar et al., 2008;Skodlar and Parnas, 2010). These factors may increase the risk of suicidality directly or indirectly by increasing the level of depression.

Neurocognitive function in schizophrenia

Neurocognitive impairments are widely documented as important features of schizophrenia, and have potential implication for prognosis, real-world functioning and long term outcome (Keefe et al., 2006;Heinrichs, 2005).

About 85% of patients with schizophrenia have neurocognitive impairments, defined as performance 1 standard deviation below healthy controls in more than two domains (Reichenberg et al., 2009). Even patients who perform within the normal range on neuropsychological tests seem to be impaired relative to their estimated intellectual function (Reichenberg et al., 2005).

Neurocognitive impairments have been documented in both early onset populations (Holmen et al., 2010), first episode patients (Bilder et al., 2000; Binder et al., 1998; Mesholam-Gately et al., 2009; Rund et al., 2004), and late phases of the disorder, as well as before illness onset (Caspi et al., 2003). These impairments are also present in high risk populations and in unaffected first degree relatives (Erlenmeyer-Kimling et al., 2000; Cole et al., 2011; Staal et al., 2000).

Neurocognitive impairments are found across most domains in the majority of patients with schizophrenia (Bowie and Harvey, 2006). Impairments in the domains of verbal learning and memory, psychomotor speed, and attention have been specifically reported in first episode schizophrenia spectrum disorders (Skelley et al., 2008; Townsend and Norman, 2004).

Most previous studies have not found any strong associations between positive psychotic symptoms and neurocognitive deficits (Nieuwenstein et al., 2001), but there are some inconsistency in the findings. However, even when strong associations between symptom severity and neurocognitive function are evident, even schizophrenia patients with low severity of such symptoms exhibit profound neurocognitive impairment. There is some consistency in documenting an association between negative symptoms and severity of neurocognitive deficits, particular deficits in executive functions (Reichenberg, 2010).

Some studies have shown that neurocognitive impairment may be more severe in males than in females with schizophrenia (Heinrichs, 2005).

Because neurocognitive impairments remain stable over the course of illness, and do not appear to be secondary to symptoms or medications (Rund et al., 2004; Nieuwenstein et al., 2001), they are increasingly considered as endophenotypic traits of schizophrenia (Gur et al., 2007).

2. AIMS

The overall aim of the present study was to estimate the level of SDs, measured by the EASE, in patients with first episode psychosis, and to investigate the relationship between the rate of self-disturbances and other patient characteristics.

In the first paper, the aim was to assess the inter-rater reliability of the EASE.

In the second paper, the aim was to investigate whether SDs, could discriminate between schizophrenia spectrum psychoses, bipolar psychoses and other psychotic disorders usually classified outside of the (narrow) schizophrenia spectrum (delusional disorder, brief psychotic disorder and psychosis NOS) in the early phase of the disorder, and thus improve differential diagnostics.

In the third paper, the aim was to investigate whether suicidality in early phases of schizophrenia may be partly motivated by SDs, either directly or indirectly through increased feelings of depression.

In the fourth paper, the aim was to investigate the relationships between SDs and neurocognitive test performance in the early phase of schizophrenia

We posed the following questions:

1.

Does the EASE provide a reliable and internally consistent clinical tool for the assessment of subjective experience in first-treatment schizophrenia patients?

2.

Can SDs discriminate between schizophrenia spectrum psychoses (schizophrenia, schizophreniform disorder and schizoaffective disorder) (SZ), bipolar disorders (bipolar disorder I and NOS) (BD) and other psychotic disorders usually classified outside of the (narrow) schizophrenia spectrum (delusional disorder, brief psychotic disorder and psychosis NOS) (OP) in the early phase of the treated course of the disorder?

3.

Is current suicidality related to SDs in first-treatment schizophrenia patients, and how is the relationship between suicidality, depression and self-disorders in these patients?

4.

How are the relationships between neurocognitive dysfunctions and SDs in the early phase of schizophrenia?

3.METHODS

Design

The present study has a cross-sectional, naturalistic design. It is an independent part of the TOP (Thematic Organized Psychosis) research study, and it is supported by the Network for Early Stage Psychosis Research (Helse SørØst Kjernekompetansemiljø). The TOP study is a large, multisite research study carried out by the University of Oslo, aiming at gaining more

knowledge about the underlying pathophysiological mechanisms of psychoses. The main diagnoses for the patients included in the TOP study are schizophrenia spectrum disorders and bipolar spectrum disorders. The TOP study is approved by the Regional Committee for Medical Research Ethics and the Norwegian Data Inspectorate.

The current study was carried out in the Department of psychosis and rehabilitation at Sykehuset Innlandet HF (SIHF) in close cooperation with other research projects on psychotic disorders within SIHF, and the research milieu on self-disturbances at Sykehuset Buskerud HF (SBHF).

Study population

The clinical sample in the current study is recruited from a naturalistic clinical setting; namely all treatment facilities in two neighbouring Norwegian counties (Hedmark and Oppland) with a county-wide population of 375.000 people. Patients were recruited from altogether 19 treatment units in Innlandet Hospital Trust (SIHF). All the patients were Norwegian citizens. Inclusion criteria were: 1: a DSM-IV diagnosis of schizophrenia (schizophrenia, schizoaffective disorder, schizophreniform disorder), bipolar disorder (bipolar I and bipolar NOS), delusional disorder, brief psychosis or psychosis not otherwise specified, 2: being consecutive in- or outpatient referred to SIHF for first adequate treatment (FAT), 3: being between 18 to 65 years old.

Exclusion criteria were the presence of head injury with neurological complications, neurological disorder and mental retardation, $IQ < 70$ (Wechsler Abbreviated Scale of Intelligence (WASI) (Wechsler, 2007; Wechsler, 2003)). Patients with concurrent substance use disorders were included, but had to demonstrate at least one month without substance use, or clear signs that the psychotic disorder had started before the onset of significant substance use (i.e. did not meet the criteria for substance induced psychotic disorder). Coming to FAT was defined as not having

previously received adequate antipsychotic medication (adequate doses for 12 weeks or until remission). Some of the patients had not initiated first treatment at the time of inclusion.

To enhance statistical power, we also included 18 patients consecutively enrolled in a closely related ongoing study on young psychosis patients born in 1985/86 (by Unni Bratlien, M.D.).

They met the same inclusion and exclusion criteria except for the strict definition of first treatment.

During 2008 and 2009 we recruited a total of 100 patients. Six patients were excluded for diagnostic reasons, one had a mental retardation, and two refused the EASE interviews, giving a final sample of 91 patients who completed the full protocol including the EASE interview, 73 FAT patients and 18 patients from the age cohort (1985/86).

In the first paper, we used 25 randomly drawn videotaped interviews from the full sample. In the second paper we used the whole sample (n=91) because we looked for differences between diagnostic groups. In the third paper, we only included FATs with a diagnosis of SZ (n=49), while in the fourth paper we included all the patients with SZ (n=57).

Table 1. Sampling procedure for the four individual substudies

Data collection jan 08-des 09	Selection criteria	Paper	Substudy samples
TOP database sample n.91	Randomly drawn videotaped EASE interviews	1	25
First adequate treatment n73, Birth cohort 18	Schizophrenia spectrum disorder	2	91
Schizophrenia spectrum disorder n.57	Bipolar disorder		
Bipolar disorder n.21	Other psychoses		
Other psychoses n.13	Schizophrenia spectrum disorder	3	49
	First adequate treatment		
	Schizophrenia spectrum disorder	4	57

Assessments

Clinical assessments

Diagnoses were ascertained by two experienced psychiatrists (EH and UB) using the Structural Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (SCID-IV) (1994). Symptom severity and function were assessed using standard psychiatric measures including the Structured Clinical Interview for the Positive and Negative Syndrome Scale (SCI-PANSS) (Kay et al., 1987). Calgary Depression Scale for Schizophrenia (CDSS) (Addington et al., 1990), Young Mania Rating Scale (YMRS) (Young et al., 1978). Global Assessment of Functioning - Split Version (GAF-S) (Endicott et al., 1976; Pedersen et al., 2007). We also registered the following data: duration of untreated psychosis (DUP), present medical treatment, first hospitalisation for a psychiatric problem, earlier symptoms and treatment and parasuicidal episodes, and family history of psychiatric illnesses.

Assessments of self-disorders

SDs were assessed according to the EASE manual (Parnas et al., 2005b). The EASE is a symptom checklist supporting the semi-structured exploration and classification of experiential anomalies that may be considered disorders of basic or core self-awareness. It is divided into five domains, as described in the introduction. The EASE is not structured as an interview, but rather as a detailed descriptive and exemplified manual. Therefore we decided to make a supplementary Norwegian semi-structured interview guide, intimately based on the manual. This work was done by my main supervisor, Paul Møller, one of the main authors of the EASE, in collaboration with me. Using this interview guide, I conducted 8 videotaped pilot-interviews prior to the ordinary inclusion. The final interview guide, used in the current study, was adjusted after experiences we

did with these pilot-interviews. All patients were asked for consent to videotape the EASE interview, 65 % of the patients gave their consent, and their interviews were videotaped. The interview guide provides several alternative question probes for each item. Because this was a research study, I went through the items one by one, from the beginning to the end of the interview guide. I started with the first question probe for each item and waited for response before asking supplementary questions. Even though we asked questions like: "Have you ever felt as if you were somebody else?" such experiences cannot be assessed by affirmative or denying responses alone. The patients were stimulated optimally to give at least one example or a description of the experience to assure that they had understood the question and that they really had experienced the actual phenomenon I asked for. At the best, they were able to give spontaneous examples or descriptions of the experience. The answers were scored on a 5-point Likert scale:

0=never present. 1=questionably present. 2=mild level; irregularly, but at least 3 times, no subjective distress. 3=moderate level; daily for one week two times during one year, or frequent for one year, may give subjective distress. 4= serious level; almost daily during two weeks recently, usually subjective distress and functional decline.

For the purpose of the analyses in this study, the 0-4 scores were dichotomized into 0 and 1 (absent or questionably present) vs. 2, 3, and 4 (definitely present, all severity levels).

In this study, all the EASE interviews were conducted by E.H. Each interview took 30-90 minutes. Because 65 % of the interviews were videotaped, this also gave me the opportunity to look through the interviews once more if I was unsure about some scores.

Neurocognitive assessments

The assessments were performed by clinical psychologists. They were trained and supervised by Merete Øie, a member of the research group and an experienced neuropsychologist. All subjects were tested individually but received the tests in the same fixed order. Total time for all assessments was about 3 hours, including breaks. Premorbid IQ was assessed with a Norwegian Research version of the National Adult Reading Test (NART) (Crawford et al., 2001; Vaskinn and Sundet, 2001) and current estimated IQ with WASI (Wechsler, 2003; Wechsler, 2007). The other tests cover domains shown to be sensitive to the neurocognitive dysfunction in psychosis (Green et al., 2004) :

Psychomotor speed: Digit Symbol from WAIS-III (Wechsler, 1997). The task is to fill in blank spaces with the symbol that is paired to the number above the blank space as quickly as possible for 120 seconds. The score is the number of squares filled in correctly.

Working memory: Letter Number Span from WAIS-III (Wechsler, 1997) This test consists of six items. Each contains three trails with the same number of digits and letters. The examinee reads each trail, and the patient is asked to recall the letters in alphabetical order and the numbers in ascending order. This task is sensitive to auditory working memory. Outcome is total correct recalled trails.

Verbal memory: Logical Memory Test from the Wechsler Memory Scale [WMS] III (Wechsler et al., 2008). This is a verbal test assessing immediate and delayed memory for two short stories orally presented. Immediate memory was used here.

Visual memory: Rey-Osterrieth Complex Figure Test (Meyers and Meyers, 1995). The subject observes a complex geometric figure for 30 seconds and then reproduces it from memory, immediately and after a brief delay (20 min) without prompting. Delayed memory of the figure was used here.

Executive functions: The Colour-Word Interference subtests from the Delis-Kaplan Executive Function System (D-KEFS) (Delis et al., 2001). This test includes four conditions: Colour Naming, Word Reading, Inhibition, and Inhibition/Switching. In the first condition the subject has to name different colors, before reading the printed words of these colors in the second condition. In the third condition the subject need to inhibit this overlearned verbal response when naming the dissonant ink colors in which the words are printed. In the fourth condition the subject is asked to switch back and forth between naming the dissonant ink colors and reading the words. Executive functions used in the present study were inhibition (3. condition) and cognitive flexibility (4. condition), and completion time in seconds was examined. Standard scores or T-scores (Rey) according to norms were used for all tests.

For both the clinical assessments, the assessments of SDs and the neurocognitive assessments, the patients did not have to be in remission, but were required not be so overtly psychotic or have so disordered cognition that they had problems participating in a lengthy interview or in understanding the nature of the informed consent. All participants gave written, informed consent to participate. The study was approved by the Regional Committee for Medical Research Ethics and the Norwegian Data Inspectorate.

Statistical analyses

All analyses were performed with the statistical package SPSS, version 15.0. All tests were 2-tailed, and limits for significance was set to the 0.05 level. In all the studies, mean and standard deviations are reported for continuous variables and percentages for categorical variables. Since DUP had a markedly skewed distribution, median and range values are reported and a transformation into its natural logarithm was used in parametric analyses. Correlations between

variables were explored by Spearman or Pearson rank correlation according to type of data.

Independent t-tests, Welch-weighted analysis of variance or Mann-Whitney U tests (dependent on the distribution of data) were used to investigate group differences for continuous data, while categorical variables were analysed with Chi-square analysis.

The reliability of the EASE manual was assessed by calculating Cohen's kappa for the agreement between the two raters , and Cronbach's alpha for the internal consistency.

In the second paper, the predictive value of the variables were explored by binary multiple logistic regression analyses.

In the third paper, regression analyses were used to assess the independent association between clinical characteristics, and also for follow-up analyses of the effect of possible confounders of their relationship. The results were examined for effect of outliers and influential observations. We used the Sobel test to evaluate mediation.

In the forth paper, spearman's rank order correlation analyses were used to investigate possible associations between different neurocognitive measures and SDs (EASE total score), and linear regression analyses were used to explore if there were possible confounders of the relationship between SDs and neurocognitive measures.

More thorough descriptions of the statistical analyses are presented in the papers.

4. SUMMARY OF PAPERS

Paper I

Examination of anomalous Self-Experience in First-Episode Psychosis: Inter-Rater

Reliability

Background

The growing research focus on early detection of schizophrenia has fostered an increasing interest in the nonpsychotic experiential anomalies that may antedate schizophrenia spectrum disorders and assist early differential diagnosis. The Examination of Anomalous Self-Experience (EASE) is a phenomenologically-inspired checklist, specifically designed to support the comprehensive assessment of these characteristic subjective experiences.

Aim

To assess the inter-rater reliability of the EASE.

Sampling and Methods

Twenty-five patients referred to FAT for a psychosis were interviewed with the EASE. Video-recorded interviews were blindly re-evaluated. Internal consistency, overall inter-rater correlation and item inter-rater agreement (Cohen's kappa) were estimated

Results

The EASE showed good to excellent internal consistency across the two raters (Cronbach's alpha above 0.87) and an overall inter-rater correlation above 0.80 (Spearman's rho, $p < 0.001$). The average kappa of the EASE was 0.65, ranging from 0.51 to 0.73 over the five domains. Kappa values at an item level were very good in nine items, good in twenty items, moderate in eleven items and fair in four items. *Conclusion.* The EASE provides a reliable and internally consistent clinical tool for the assessment of subjective experience in patients coming to FAT for psychosis,

suggesting that this instrument could usefully supplement standard clinical assessments during the onset phase of psychosis.

Paper II

Selective aggregation of self-disorders in first treatment DSM-IV schizophrenia spectrum disorders

Background

Converging evidence indicates that Self-disorders (SDs) selectively aggregate in schizophrenia spectrum conditions.

Aim

To test the discriminatory power of SDs with respect to schizophrenia and non-schizophrenia spectrum psychosis at first treatment contact.

Method

SDs were assessed in 91 patients referred for first treatment by the Examination of Anomalous Self-Experience (EASE) instrument. Diagnoses, symptoms severity, and function were assessed using the SCID-IV, Structured Clinical Interview for the Positive and Negative Syndrome Scale (SCI-PANSS), Calgary Depression Scale for Schizophrenia (CDSS), Young Mania Rating Scale (YMRS), and Global Assessment of Functioning - Split Version (GAF-S).

Results

EASE total score critically discriminated between schizophrenia, bipolar psychosis, and other psychoses. The EASE total score was the only clinical measure that showed a significant and robust association with the diagnosis of schizophrenia. *Conclusion:* Systematic exploration of anomalous self-experiences could improve differential diagnosis in first treatment patients.

Paper III

The association between anomalous self-experience and suicidality in first episode schizophrenia seems mediated by depression

Background

A recent hypothesis is that suicidality in schizophrenia may be linked to the patients' altered basic self-awareness or sense of self, termed self-disorders (SDs).

Aim

To investigate whether SDs in first episode schizophrenia spectrum disorders are related to suicidality and whether this relationship is independent of or mediated by depression or other standard clinical measures..

Method

SDs were assessed in 49 patients with first-episode schizophrenia by means of the Examination of Anomalous Self-Experience (EASE) instrument. Symptoms severity and functioning were assessed using the Structured Clinical Interview for the Positive and Negative Syndrome Scale (SCI-PANSS), Calgary Depression Scale for Schizophrenia (CDSS), and Global Assessment of Functioning - Split Version (GAF-S). Suicidality was measured by CDSS item 8.

Results

Analyses detected a significant association between current suicidality, current depression and SDs as measured by the EASE. The effect of SDs on suicidal ideation appeared to be mediated by depression.

Conclusion

The interaction between anomalous self-experiences and depression could be a rational clinical target for the prevention of suicidality in the early phases of schizophrenia, and supports the rationale for including assessment of SDs in early intervention efforts.

Paper IV

The Relationship between Self-Disorders and Neurocognitive Dysfunction in Schizophrenia

Background

Neurocognitive deficits and self-disorders (i.e altered basic self-awareness or - sense of self) have both been suggested as fundamental trait features of schizophrenia. However, no study till now has investigated the relationship between these two core features.

Aim

To investigate the relationship between self-disorders and neurocognitive performance in patients with schizophrenia.

Method

Self-disorders were assessed in 57 patients in the early phase of schizophrenia by means of the Examination of Anomalous Self-Experience (EASE) instrument. The neurocognitive assessments included measures of psychomotor speed, working memory, executive- and memory functions.

Results

There were few associations between self-disorders and neurocognitive impairments. However, high levels of SDs were significantly associated with impaired verbal memory.

Conclusion

The reason for the general lack of associations between self-disorders and neurocognition could be that they represent different basic features of the illness. Verbal memory may however be linked to deficits in the patients' ability to comprehend, direct, remember and reason about their thoughts, functions that are intimately related to several aspects of the sense of self.

5. DISCUSSION

Discussion of the Main findings

Interrater Reliability

It has been questioned whether “soft” subjective experienced like those in the EASE can be assessed reliably, pointing to the inevitable fleeting and fluctuating nature of these phenomena. Doing such investigations during psychosis might do this challenge even greater. There is however growing evidence that reliable assessment is fully achievable. As part of the present study, we found good to excellent internal consistency (Cronbach’s > 0.87), high interrater correlation (> 0.80) along with satisfactory overall kappa (0.65; 0.51-0.73) of the EASE total scores. In addition, two yet unpublished studies has shown an acceptable interrater correlation of the EASE, one for prodromal patients by B. Nelson and colleagues (personal comm.) and one for first-admitted patients by J. Parnas and colleagues (personal comm.). Together, this supports the applicability of the EASE for both clinical and research purposes. Furthermore, a significant part of the EASE overlaps with the BSABS, which has previously demonstrated good interrater reliability (Vollmer-Larsen et al., 2007). Taken together this indicates that high levels of clinical reliability are achievable by a guided, phenomenologically inspired assessment of the patient’s experience.

Self disorders in early psychoses

In this study we found that SDs, as measured by the EASE, discriminate between patients with SZ versus BD or OP in patients referred to FAT for psychosis. The EASE total score showed a significant and robust association with the diagnosis of SZ as opposed to all the other clinical measures (e.g. PANSS all subscales, YMRS, CDSS and GAF-function score). This indicates that

SDs separate psychotic schizophrenia spectrum phenotypes from non-schizophrenia spectrum psychosis.

The results in the current study are consistent with previous findings, showing a selective aggregation of SDs in schizophrenia spectrum conditions. Two qualitative studies conducted independently, but almost at the same time revealed that SDs were prominent in the prodromal phase of schizophrenia (Parnas et al., 1998; Møller and Husby, 2000). Earlier studies are non-EASE based, but have used items from the BSABS to measure SDs. Among these, one study on 151 first-admitted patients with different psychiatric disorders showed that SDs were more prominent among patients with schizophrenia spectrum conditions (including schizotypal disorder) (Handest and Parnas, 2005). Another study on 44 patients with residual schizophrenia or a psychotic bipolar disorder in remission revealed that certain anomalies of subjective experience aggregated significantly in schizophrenia (Parnas et al., 2003), and a third study on a nonpsychotic genetically high risk population (n=218) showed that SDs were associated with increasing schizotypal phenotypic expressivity (Raballo and Parnas, 2010). Finally, a study, which included 305 subjects divided into four groups (schizophrenia, schizotypal disorder, other mental illnesses and healthy controls), showed a specific aggregation of SDs in schizophrenia spectrum conditions (Raballo et al., 2011).

Also, the distribution and levels of SDs that were found in the current study are in accordance with those found in earlier, non-EASE-based studies (Parnas et al., 2003; Parnas et al., 2005a; Raballo and Parnas, 2010). Taken together, SDs might be a candidate phenotypic marker of schizophrenia.

Self disorders, depression and suicidality in schizophrenia

The study strongly supports the role of SDs in the development of suicidal ideation and behaviour. We revealed a strong association between current suicidality and SDs in patients with schizophrenia coming to their FAT.

This association between SDs and suicidality is in line with previous findings. Recently, one qualitative study of 19 patients with chronic schizophrenia showed that feelings of profound solitude, inferiority and sense of fundamental inability to relate to others, were associated with suicidality (Skodlar et al., 2008). A later expansion of this sample (n=25) measuring SDs with the EASE showed that suicidality was associated with SDs, mediated through these specific feelings (Skodlar and Parnas, 2010).

We also found a strong correlation between SDs and depression, and between depression and suicidality, indicating that the effect of SDs on suicidal ideation is mediated by depression.

The relationship between subjective experiences and depression in psychotic disorders has been studied to a limited extent, and as far as we know, there are no previous studies exploring the relationship between SDs, as measured by the EASE, and depression. However, in a cross-sectional study, which involved 161 patients with chronic schizophrenia, they found significant associations between important facets of depression (i.e. depression, hopelessness and ideas of reference) and Basic Symptoms (Maggini and Raballo, 2006). Furthermore, in a previous study of 50 patients with chronic schizophrenia, it was found that awareness of psychological deficits measured by the Subjective Experience of Deficits in Schizophrenia (SEDS) was associated with depression. Awareness of these deficits was present before, during and after the depressive period, indicating that this was not merely an epiphenomenon based on depressed patients being more acutely aware of their deficits (Liddle et al., 1993). Clinical experience also indicates that

SZ is highly associated depression, while depressive illness per se is not associated with SZ. So, the direction SDs to depression seems more likely than depression to SDs.

Self disorders and neurocognitive deficits in schizophrenia

Our main finding concerning neurocognition is a general lack of associations between SDs and neurocognition. We found, however, that higher levels of SDs were associated with impaired verbal immediate memory. Except for that, no significant association between SDs and neurocognitive impairment such as working memory, executive function, psychomotor speed or visual memory were found.

There are no previous studies that have examined the relationship between SDs, measured by the EASE, and neurocognitive impairments. There are, however, a few small studies which have investigated the relationship between Basic symptoms (BS) and neurocognitive deficits. BS are subclinical disturbances in drive, affect, thinking, speech, (body) perception, motor action, central vegetative functions, and stress tolerance (Schultze-Lutter, 2009). Our study is in line with two previous studies showing no significant association between SDs and neurocognitive impairment. One study on 50 outpatients with chronic schizophrenia, reported BS (measured with the Frankfurt Complaint Questionnaire (FCQ)) to be unrelated to measures of executive functioning (Zanella and Huguelet, 2001). Another study on prodromal patients focusing on neurocognition and selected BSs in the SPI-A (Schultze-Lutter et al., 2007a), did also not find any significant correlation between the subjective disturbances and objective neurocognitive function (pattern recognition, attention, working memory, verbal and visual memory, psychomotor speed, and executive functions) (Schultze-Lutter et al., 2007b). A third study of 32 inpatients with schizophrenia, consecutively admitted due to a recrudescence of their

symptomatology, however, revealed associations between BSs (measured with the FCQ) and impairments of executive functioning, psychomotor speed and subtests of Wechsler Adult Intelligence Scale (WAIS) (Cuesta et al., 1996). These three studies, however, focused on different patient groups, SDs were not measured by the EASE but instead BSs were measured, and by different scales (FCQ and SPI-A).

A possible explanation for the general lack of associations between SDs and neurocognition is that SDs and these objective neurocognitive functions are different basic expressions of the illness, and that different networks of the brain are involved. The neurocognitive functions measured in the present study are associated with functions mediated by dorsolateral prefrontal cortex, temporal- and unspecific subcortical regions of the brain. The neurocognitive test situation is structured with little affective- and somatosensory salience. However, in contrast to the neurocognitive tests used in this study, the questions asked in the EASE have focus on subjective experiences that are present and relevant in almost all everyday situations, involving somatosensory and affective processes, interacting with neurocognition. These processes have been associated with activation of other networks of the brain involving the thalamus, orbito-frontal cortex, the limbic system and several distinct somatosensory cortices in the insular and parietal regions (Damasio, 1994).

SDs were, however, significantly correlated with verbal immediate memory. The self is a dynamic structure with a set of multidimensional representations stored in memory. New information and new thoughts are processed in relation with preexisting self-knowledge. The verbal memory test used in the current study requires rapid cognitive processing of incoming verbal information and efficient organization for accurate recall. One possibility is that deficits in verbal memory may cause deficits in the ability to comprehend, direct, remember and reason

about one's own thoughts and self-knowledge, functions that can be seen as related to several aspects of SDs, or the sense of self. However, it might as well be the other way around, that impaired SDs is the primary deficit; causing impaired verbal memory. This is, however, speculations, and more research is necessary to explore the relationship between verbal memory and SDs.

We did not find any significant associations between PANSS scores and verbal memory or SDs, thus there were no indications that the relationship between SDs and verbal memory was mediated through clinical symptoms. Theoretically, the lack of association between SDs and neurocognition could be a result of gender differences. However, our results showed no gender differences in SDs, estimated IQ or verbal memory, and follow-up analyses also indicated that gender was not a confounder.

Discussion of methodological issues

Sample representativity

The study population is recruited from a naturalistic clinical setting, involving all treatment facilities in two neighbouring Norwegian counties and including unselected consecutive in- or outpatients referred to FAT for a psychotic disorder in a defined time period. The Norwegian mental health care offers public mental health care to all individuals with mental illness within a given catchment area. Because of the absence of private mental health care in Norway, the sample is not biased for socioeconomic class. We thus assume that the sample is close to the total treated incidence for this time period i.e. highly representative.

To enhance statistical power, we also included 18 patients consecutively enrolled in a closely related ongoing study of young psychosis patients born in 1985/86 (by Unni Bratlien, M.D.).

They met the same inclusion and exclusion criteria except for the strict definition of first treatment. They were, however, in an early phase of their treatment course, with an even shorter mean duration of untreated psychosis (DUP) than the strict first treatment patients.

All the patients were included in the early phase of the treated course of the disorder, thereby minimizing potential confounding effects such as selection of non-responders, chronicity and substance use or medication use, that might impact on the assessment of clinical symptoms and neurocognitive functioning.

Our study was the first study on first-episode psychoses in the current catchment area. We recruited from altogether 19 different units in this area, and only a few of these units had early treatment programs for psychoses. A considerable amount of the participants included in the current study had previously been in the treatment system, but not diagnosed with a psychotic disorder, and therefore not treated for such. This could be a possible explanation for the relatively long DUP in this sample

Validity and reliability of assessments

Assessment of SDs with the EASE

SDs were in this study assessed with the EASE manual, a comprehensive checklist for SDs. This is a descriptive manual with examples, not a semi-structured interview, therefore we constructed a Norwegian interview guide (see Methods).

The EASE is a relatively new instrument and until now, not widely used. Therefore the IRR for the EASE items was examined on the basis of 25 randomly drawn videotaped interviews and examined by PM, who was blind to diagnostic and other clinical information. The IRR was found to be very good with an overall inter-rater correlation of the EASE total score above 0.80 (Spearman's coefficient, $p < 0.001$), and kappa = 0.65. The internal consistency of the EASE

scale was found to be very good across the two raters with a Cronbach's alpha for the whole scale above 0.85 (Møller et al., 2011).

False negatives. Some patients may come out with false low levels of SDs. SDs have a pre-reflexive quality, and therefore it can be difficult to get a grasp of it. SDs themselves also undermine the patients ability to express themselves. To overcome this, I asked more than one question for every single item if the patient did not respond convincingly to the first question. I could also come up with examples to see if the patient recognized this and told me whether he had similar experiences. Several items in the EASE have partial overlap, inevitable from the "gestalt" nature of the phenomena, so if a person has a positive answer to one item, it is likely that he also has a positive answer to related items. One example is "diminished sense of basic self" and "distorted first-person perspective" which overlaps clinically at a descriptive level because they are conceptually and phenomenologically related.

False positives. There is also a risk for false positive scores. Some may give a positive response to a question, even though the quality of the reported experience is not what we are looking for. To avoid scoring this as present, I always asked for examples or closer descriptions of the actual experience, and I never considered a simple "yes" enough to score the item.

Therefore, to minimize false positive or false negative scores, the interviewer must have detailed knowledge of phenomenological and descriptive psychopathology in general and of schizophrenia spectrum conditions in particular. One also has to be familiar with the EASE manual and the EASE interview, to be sure about targeting not only the plain content, but also structural aspects of self-consciousness.

Assessment of neurocognition

The comprehensive neuropsychological test battery used in the current study cover domains shown to be sensitive to the neurocognitive dysfunction in psychosis (Green et al., 2004).

It was conducted by psychologists blind to information about the EASE score, trained in the test battery and supervised by an experienced neuropsychologist (MØ). To assure valid assessment of neurocognitive test performance all participants had to score 15 or above on the forced recognition trial of the California Verbal Learning Test (CVLT II).

Clinical assessments

The other instruments used in this study (SCID, SCI-PANSS, CDSS, YMRS, GAF-split version) are widely used and have their reliability and validity well documented.

The two investigators who performed the clinical assessments in the current study completed the TOP study's training and reliability program. The SCID training was based on the UCLA training program (Ventura et al., 1998) and was supervised by UCLA. For DSM-IV diagnostics, mean overall kappa for the standard diagnosis of training videos was 0.77, and mean overall kappa for a randomly drawn subset of the present study patients was also 0.77 (95% CI 0.60-0.94). Inter-rater reliability (Intra Class Coefficient) (ICC) 1.1 for the different psychometric scales was: PANSS positive subscale 0.82 (95% CI 0.66-0.94), PANSS negative subscale 0.76 (95% CI 0.58-0.93), PANSS general subscale 0.73 (95% CI 0.54-0.90), and GAF-F 0.85 (95% CI 0.76-0.92). These numbers are based on the scores for the whole TOP study group. EH and UB are a part of this group.

Strengths

This is a naturalistic study with a study population that is highly representative (see "sample representativity"). The recruitment, in-depth structured clinical interviews and clinical measures were conducted by experienced psychiatrists (UB and EH), who knew the area and the facilities well.

Limitations

The current study has a cross-sectional design. It shows whether SDs are present when someone has reached treatment, but it does not show whether these are risk factors, genetic markers or consequences of illness. However, our results combined with previous studies suggest that SDs reflect non-psychotic, trait-like distortions of self-awareness which also has been shown in prodromal studies to antedate the development of clearly psychotic experiences (Møller and Husby, 2000; Parnas et al., 1998).

The cross-sectional design also makes it difficult to clarify the causal direction between depression and SDs.

On the basis of the cross-sectional design, it is also not possible to show whether the SDs may be the primary deficit which affects verbal memory, or whether it may be the other way around. Some participants were not in full remission at the time of neurocognitive testing, which may have confounded the results with potential effects of psychotic symptoms. However, all participants scored 15 or above on the forced recognition trial of the CVLT II, indicating adequate and valid test performance.

It was not possible to be blind to diagnostic information during the EASE interviews because I, during inclusion procedures, also administered the SCID for one half of the sample. Under all circumstances, and because of epistemological reasons, it is impossible not to form an assumption about a subject's diagnosis during the EASE exploration (i.e. a clinically experienced psychiatrist would not be able to avoid making diagnostic assumptions during the EASE, since the assessment per se brings to the foreground the very structure of the patients' mode of experiencing). Still, as a precaution, we performed an ANOVA with Bonferroni's post hoc tests comparing ratings across the two raters, and did not detect any significant differences in

the EASE total scores for each diagnostic group as a function of who did the SCID interview (EH or UB).

Implications.

Our results, combined with previous findings of an aggregation of SDs in schizophrenia spectrum disorders (Parnas et al., 2005a; Parnas et al., 2003; Raballo and Parnas, 2010; Møller and Husby, 2000; Parnas et al., 1998), indicate that SDs might be a candidate phenotypic marker of schizophrenia.

SDs and the diagnostic criteria for schizophrenia

Historically, profoundly altered self-experience has been known as a core feature of schizophrenia (Parnas and Handest, 2003). Despite this, the diagnosis of schizophrenia is based on diagnostic criteria (in ICD-10 or DSM-IV), which do not include this altered self-experience. Furthermore, the DSM-IV and ICD-10 criteria have an indistinct, unspecific and overlapping nature which does not differentiate well between schizophrenia, psychotic bipolar disorder and other psychoses in the early stages. In the current study we found that EASE total score differentiated significantly and strongly between schizophrenia and other psychotic disorders (including bipolar I and bipolar NOS psychosis) in the early stages of the diseases, suggesting that the assessment of the SDs is promising to assist early differential diagnosis. Our results also confirm that high levels of clinical reliability are achievable by a guided, phenomenologically inspired assessment of the patient's experience. Therefore, one might also consider whether SDs should be included in the diagnostic criteria for schizophrenia in future diagnostic systems. Furthermore, this perspective should have implications for therapeutic interventions as it gives meaning to the patients through making strange and scaring experiences more understandable and

thus possible to talk about. Knowledge about SDs also makes it easier for clinicians to understand what patients are trying to communicate.

The role of SDs in the development of suicidal behaviour

Suicidal ideation and behaviour and subsequent high risk of suicide are major complications in schizophrenia. However, risk factors, identified until now, have a low predictive power, making it difficult to initiate targeted suicide prevention in clinical settings.

In the current study, we found a clear association between current suicidality and SDs, which appears to be mediated by depression. These findings strongly support the role of SDs in the development of suicidal ideation and behaviour in this patient group and contribute to the clinical understanding of suicidality in schizophrenia. Therefore, the interaction between self-disorders and depression could be a rational clinical target for the prevention of suicidality in the early phases of schizophrenia, and for early intervention efforts in general.

The relation between SDs and neurocognitive deficits

Both SDs and neurocognitive deficits have been suggested as fundamental trait features of schizophrenia.

The general lack of association between SDs and neurocognitive impairments in the current study, except for verbal memory, indicates that these problems are different basic expressions of the illness. Therefore, early assessment of neurocognitive impairments as well as SDs is important for therapeutic and diagnostic reasons.

Future research

More studies. The EASE, used in this study, is a relatively new instrument. However, it provides a reliable and internally consistent clinical tool for the assessment of subjective experience.

Therefore we should have more studies, using the EASE, to explore the relationship between SDs and other patient characteristics. As far as we know, there are no previous studies on the relationship between SDs (measured by the EASE) and neurocognitive impairments. Therefore, more and larger studies are required to further clarify these issues. We also need to assess SDs in the general population.

Longitudinal studies. In the current study, we could only assess the discriminatory power of SDs in a cross-sectional design. Thus, longitudinal studies on UHR patients, other at-risk groups, first episode patients and patients after recovery are required to further clarify the SD's early course and predictive potential. We also need longitudinal studies to explore the relationship between SDs, depression and suicidality.

Treatment response. There are currently no empirical data regarding the treatment response of SDs, and their trajectory during recovery is still unexplored, therefore we also need future studies to explore these issues.

6. CONCLUSION

1. The EASE provides a reliable and internally consistent clinical tool for the assessment of subjective experience in patients coming to FAT for psychosis.
2. In the current study SDs, as measured by the EASE, discriminate between patients with SZ diagnosis versus BD or OP in patients referred to FAT for psychosis.

3. We found a clear association between current suicidality and SDs, which appears to be mediated by depression.

4. In our study, the level of SDs is significantly associated with verbal memory but not with working memory, executive function, psychomotor speed or visual memory in patients with early phase schizophrenia.

Taken together these studies have shown that SDs are core symptoms of schizophrenia and describe a gestalt more than separate symptoms.

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8. APPENDIX

Appendix 1: EASE Item Key List

Domain 1. Cognition and stream of consciousness

- 1.1 Thought interference
- 1.2 Loss of thought ipseity
- 1.3 Thought pressure
- 1.4 Thought block
 - 1.4.1 Subtype 1: blocking
 - 1.4.2 Subtype 2: fading
 - 1.4.3 Subtype 3: combination
- 1.5 Silent thought echo
- 1.6 Ruminations-obsessions
 - 1.6.1 Subtype 1: pure rumination
 - 1.6.2 Subtype 2: secondary rumination
 - 1.6.3 Subtype 3: true obsessions
 - 1.6.4 Subtype 4: pseudo-obsessions
 - 1.6.5 Subtype 5: rituals/compulsions
- 1.7 Perceptualization of inner speech or thought
 - 1.7.1 Subtype 1: internalized
 - 1.7.2 Subtype 2: equivalents
 - 1.7.3 Subtype 3: internal as first-rank symptom
 - 1.7.4 Subtype 4: external
- 1.8 Spatialization of experience
- 1.9 Ambivalence
- 1.10 Inability to discriminate modalities of intentionality
- 1.11 Disturbance of thought initiative/intentionality
- 1.12 Attentional disturbances
 - 1.12.1 Subtype 1: captivation by details
 - 1.12.2 Subtype 2: inability to split attention
- 1.13 Disorder of short-term memory
- 1.14 Disturbance of time experience
 - 1.14.1 Subtype 1: disturbance in subjective time
 - 1.14.2 Subtype 2: disturbance in the existential time (temporality)
- 1.15 Discontinuous awareness of own action
- 1.16 Discordance between expression and expressed
- 1.17 Disturbance of expressive language function

Domain 2 . Self-awareness and presence

- 2.1 Diminished sense of basic self
 - 2.1.1 Subtype 1: early in life
 - 2.1.2 Subtype 2: from adolescence
- 2.2 Distorted first-person perspective
 - 2.2.1 Subtype 1: mineness/subjecthood
 - 2.2.2 Subtype 2: experiential distance
 - 2.2.3 Subtype 3: spatialization of self

- 2.3 Psychic depersonalization (self-alienation)
 - 2.3.1 Subtype 1: melancholiform depersonalization
 - 2.3.2 Subtype 2: unspecified depersonalization
- 2.4 Diminished presence
 - 2.4.1 Subtype 1: not being affected
 - 2.4.2 Subtype 2: distance to the world
 - 2.4.3 Subtype 3: as subtype 2 plus derealization
- 2.5 Derealization
 - 2.5.1 Subtype 1: fluid global derealization
 - 2.5.2 Subtype 2: intrusive derealization
- 2.6 Hyperreflectivity; increased reflectivity
- 2.7 I-split
 - 2.7.1 Subtype 1: I-split suspected
 - 2.7.2 Subtype 2: 'as if' experience
 - 2.7.3 Subtype 3: concrete spatialized experience
 - 2.7.4 Subtype 4: delusional elaboration
- 2.8 Dissociative depersonalization
 - 2.8.1 Subtype 1: 'as if' phenomenon
 - 2.8.2 Subtype 2: dissociative visual hallucination
- 2.9 Identity confusion
- 2.10 Sense of change in relation to chronological age
- 2.11 Sense of change in relation to gender
 - 2.11.1 Subtype 1: occasional fear of being homosexual
 - 2.11.2 Subtype 2: a feeling as if being of the opposite sex
- 2.12 Loss of common sense/perplexity/lack of natural evidence
- 2.13 Anxiety
 - 2.13.1 Subtype 1: panic attacks with autonomous symptoms
 - 2.13.2 Subtype 2: psychic-mental anxiety
 - 2.13.3 Subtype 3: phobic anxiety
 - 2.13.4 Subtype 4: social anxiety
 - 2.13.5 Subtype 5: diffuse, free-floating pervasive anxiety
 - 2.13.6 Subtype 6: paranoid anxiety
- 2.14 Ontological anxiety
- 2.15 Diminished transparency of consciousness
- 2.16 Diminished initiative
- 2.17 Hypohedonia
- 2.18 Diminished vitality
 - 2.18.1 Subtype 1: state-like
 - 2.18.2 Subtype 2: trait-like

Domain 3. Bodily experiences

- 3.1 Morphological change
 - 3.1.1 Subtype 1: sensation of change
 - 3.1.2 Subtype 2: perception of change
- 3.2 Mirror-related phenomena
 - 3.2.1 Subtype 1: search for change
 - 3.2.2 Subtype 2: perception of change

- 3.2.3 Subtype 3: other phenomena
- 3.3 Somatic depersonalization (bodily estrangement)
- 3.4 Psychophysical misfit and psychophysical split
- 3.5 Bodily disintegration
- 3.6 Spatialization (objectification) of bodily experiences
- 3.7 Cenesthetic experiences
- 3.8 Motor disturbances
 - 3.8.1 Subtype 1: pseudo-movements of the body
 - 3.8.2 Subtype 2: motor interference
 - 3.8.3 Subtype 3: motor blocking
 - 3.8.4 Subtype 4: sense of motor paresis
 - 3.8.5 Subtype 5: desautomation of movement
- 3.9 Mimetic experience (Resonance between own movement and others' movements)

Domain 4. Demarcation/transitivity

- 4.1 Confusion with the other
- 4.2 Confusion with one's own specular image
- 4.3 Threatening bodily contact and feelings of fusion with another
 - 4.3.1 Subtype 1: feeling unpleasant, anxiety provoking
 - 4.3.2 Subtype 2: feeling of disappearance, annihilation
- 4.4 Passivity mood
- 4.5 Other transitivity phenomena

Domain 5. Existential reorientation

- 5.1 Primary self-reference phenomena
- 5.2 Feeling of centrality
- 5.3 Feeling as if the subject's experiential field is the only extant reality
- 5.4 'As if' feelings of extraordinary creative power, extraordinary insight into hidden dimensions of reality, or extraordinary insight into own mind or the mind of others
- 5.5 'As if' feeling that the experienced world is not truly real, existing, as if it was only somehow apparent, illusory or deceptive
- 5.6 Magical ideas linked to the subject's way of experiencing
- 5.7 Existential or intellectual change
- 5.8 Solipsistic grandiosity

Appendix 2:

EASE interview guide

(This is a Norwegian version, since there is currently no English version available.)

E A S E

Examination of Anomalous Self-Experience

Norsk intervjuguide for

Undersøkelse av forstyrret selvopplevelse ved schizofreni og relaterte lidelser

Originalpublikasjon av EASE manualen:

Parnas J, Møller P, Kircher T et al. Psychopathology 2005; 38: 236-258.

Intervjuguiden er utviklet av Paul Møller

FoU-enheten, Psykiatrisk klinikk, Sykehuset Buskerud HF, 2008

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Instruksjoner

I vanlig klinisk utredning anbefales det at EASE-intervjuet foretas som del av et sosialt eller anamnestisk intervju. Det optimale intervju er at utforskningen av de ulike selv-forstyrrelser i stor grad gjøres ved å bygge ut et slikt ordinært intervju. I løpet av intervjuet vil det naturlig dukke opp muligheter for å utforske ledd fra de ulike domeneene. I forskningssammenheng kan intervjuet brukes mer semi-strukturert, fra begynnelse til slutt, men også her er det å foretrekke at intervjuet har en åpen, ledig og dialogisk form som til dels følger pasientens fortelling. Denne fenomenologisk-kliniske metode står i en viss kontrast til den strukturerte metode. Den informasjonen vi er ute etter er ikke bare av eksplisitt (refleksiv) natur, men også implisitt (prerefleksiv), hvilket innebærer at mange av fenomenene kan være flytende og uklart artikulert hos personen, og at intervjuet skal bidra til mest mulig klargjøring.

Intervjueren må være fortrolig med manualen, kjenne definisjonene av de ulike selv-forstyrrelser, og ha et visst grunnleggende kjennskap til fenomenologisk teori. Man bør fortløpende notere viktige ord og setninger vedkommende sier, mest mulig ordrett. De spontane beskrivelsene er de viktigste. Ved overlappende fenomener skal alle relevante items skåres.

Det er ofte nødvendig å stille presiserende oppfølgingsspørsmål. Be om nærmere beskrivelse eller ett eksempel til alle items. Ja og nei er som hovedregel ikke tilstrekkelig svar. Det er et krav at personen på en eller annen måte, direkte eller indirekte, må kunne illustrere positive svar. Og illustrasjonen bør noteres. Mange av opplevelsene er det vanskelig å finne ord for. Intervjueren og den som intervjues må i fellesskap sikre en mest mulig felles forståelse av fenomenene.

Overfor pasienter som har mye tvil, kan det være nyttig å avslutte spørsmålet med følgende: - eller ikke i det hele tatt?

Et vesentlig forhold som kan lette bedømmelsen av de rapporterte opplevelser, er at de aller fleste ekte fenomener vil være forbundet med betydelige subjektive plager og vesentlig funksjonsfall. Dette bør det derfor spørres om.

For hvert item er det formulert flere spørsmål. *Start med å stille kun det første spørsmålet i formuleringen*, - og vent deretter litt på respons. Flere spørsmål etter hverandre blir lett overveldende. Ett eller flere av de påfølgende spørsmål kan benyttes hvis personen nøler eller trenger hjelp til å forstå hva det spørres etter. (Hvis han etter et par forsøk ikke synes å forstå hva du spør om, har han antakelig ikke har hatt en slik opplevelse.)

0 = aldri; **1 = tvisomt** (vagt, x1-2); **2 = mild** (uregelmessig, men minst x3, *gir ikke* subj. lidelse); **3 = moderat** (daglig i 1 uke x2 pr år ELLER hyppig i 1 år, *kan gi* subj. lidelse); **4 = alvorlig** (nesten daglig over 2 uker nylig, *gir oftest* subj. lidelse og funksjonssvikt); 9 = utilstrekkelig info

Innledning for pasienten:

”Det vi vil med denne samtalen er å utforske, beskrive og forstå så godt som mulig en del spesielle opplevelser eller oppfatninger som du muligens har nå, eller har hatt tidligere i livet ditt, og som har vært viktige for deg. Slike opplevelser er det nyttig å få beskrevet uansett om de har forekommet kortvarig, i episoder eller mer vedvarende. Det er ingen riktige eller gale svar på spørsmålene, du skal bare beskrive hvordan de er, eller var, for deg.

Hvis du svarer ja på et spørsmål vil jeg oftest be deg om å beskrive det nærmere, eller gi et eksempel, for at jeg skal være sikker på at du svarer på det jeg mener å spørre om.”

Hvis pasienten er opptatt av psykose/ikke psykose:

”Noen av de opplevelsene jeg spør om, kan hos enkelte bety en viss økt risiko for det vi kaller psykose (forklar seinere), men vi kan ikke på forhånd vite med sikkerhet hvorvidt psykose utvikler seg, eller ikke. Dette må alltid vurderes samlet – over tid – og av flere fagfolk.

Intervjuguide til E A S E

Opplysninger vedrørende Domene 1 – Tenkning og bevissthetsstrøm

Først vil jeg spørre litt om hvordan tenkningen og bevisstheten din fungerer

1.1. Har du noen gang opplevd at det helt av seg selv dukker opp *forstyrrende, nye tanker*? - som trenger seg på og blander seg inn i det du tenker på? (**Tankeinterferens**)

At du f.eks. under en samtale ikke klarer å konsentrere deg fordi tankene hele tiden veksler over til andre tema? (- som ikke har sammenheng med det du holder på med). Kan også gjelde indre bilder, innskytelser o.l.

Merk: Slike forstyrrende tanker kan også føles uvirkelige eller fremmede, tankeipseitet (1.2). De kan også øke i frekvens og gli over i tankepress (1.3). I slike tilfelle skåres begge eller alle items samtidig.

1.2. Har du noen gang opplevd at tankene i hodet ditt *ikke føles som dine egne*? – at de føles uvirkelige? - fremmede eller rare? (**Tankeipseitet**)

Som om tankene ikke tenkes *av* deg, men likevel *i* deg? Det trenger ikke være *innholdet i* tanken som føles fremmed, men hele ”det å tenke”.

1.3. Har du noen gang følt at *mange og usammenhengende* tanker kommer og går *veldig fort*, og at du ikke greier å styre det? (**Tankepress**) (Evt. indre bilder)

1.4. Har du noen gang opplevd at tankene blir borte eller blokkeres (tanketomt), *mens* du tenker?

Hvis ja: Forsvinner tankene brått eller gradvis? - blir det helt tomt etterpå? (**Tankeblokk**)

Fenomenet kan noen ganger også *observeres* i form av opphold i talestrømmen.

Subtype 1: Tanken forsvinner brått (blocking), uten ny inntrengende tanke (tanketomt)

Subtype 2: Tanken forsvinner gradvis (fading), uten ny inntrengende tanke (tanketomt)

Subtype 3: Tanken forsvinner gradvis, men *nye forstyrrende tanker* trenger inn (derfor *ikke* tanketomhet)

1.5. Har du noen gang følt at tankene på en måte blir gjentatt av seg selv? - som et slags ekko? – eller fordoblet? (**Stille tankeekko**) (NB: ikke lyd på disse ekko-tankene)

1.6. (Hvis vi ser bort fra tanker som går fort eller forstyrrer..) ..har det noen gang vært slik at du grubler så mye at det nesten ikke er mulig å stoppe det? - at du må tenke gjennom ting nesten i det uendelige? – blir liksom sittende helt fast?

(Kan også gjelde *indre bilder*, f.eks. av tidligere hendelser eller samtaler)?

(Ruminasjon /hyperrefleksjon, tvangstanker)

Merk: Dette er en vid samlegruppe som undersøkes nærmere under hyperrefleksjon 2.6.

Hvis ja: Er det en grunn til at du grubler så mye? Er det ubehagelig eller oppleves det tvert om nødvendig for deg?

Subtype 1: Primær ruminasjon. Ingen åpenbar begrunnelse for grublingen.

Subtype 2: Sekundær ruminasjon. Begrunnet i forstyrret "basic self" (2.1.), hyperrefleksivitet (2.6.), tap av common sense/ perpleksitet (2.12.), mistenksomhet, depresjon o.l.

Subtype 3: Ekte tvangstanker. Som ved OCD, ego-dystone, betydelig indre motstand.

Subtype 4: Pseudo-tvangstanker. Mer ego-syntone tanker, liten indre motstand. Ofte aggressive indre bilder, angstskapende.

Subtype 5: Hvilken som helst av subtype 1-4, pluss tvangshandlinger.

1.7. Har du noen gang følt at det er som om tankene har fått stemme eller lyd på seg (akustisk)? At du nesten kan høre dem? – eller kan du "se" tanker som tekst? Har slike ting hendt mens du f.eks. leser noe? (**Perseptualisering av tanke og indre tale [gedankenlautwerden]**)

Merk: Gjelder primært egne tanker – spesifiser hvis fremmede tanker. Ofte er slike symptomer vanskelige å datere, og oppleves egosyntont. NB: hvis pasienten frykter at andre kan høre tankene, er det et psykosesyntom, FRS.)

Subtype 1: Indre opplevelse; kommer innenfra, "høres" ikke gjennom øret, og kan ikke høres av andre.

Subtype 2: Ekvivalente fenomener, f.eks. tanker som kan sees som tekst, som blir "skrevet" samtidig som man tenker - nesten som teksten av en film.

Subtype 3: Indre opplevelse, men psykosesyntom (FRS); pasienten er redd for at andre kan høre tankene fordi de er så høylytt. (Inkluderer ikke opplevelsen av at andre kan "lese/fornemme" ens tanker [= selvhenving 5.1].)

Subtype 4: Som ytre hørselshallusinasjon eller tankeekko, som "høres" gjennom øret (fra personer eller medier)

1.8. Har det noen gang vært som om du kan *kjenne hvor i hodet* tankene er? At tanker eller følelser sitter et spesielt *sted* i hodet? Eller plassert i et bestemt forhold til andre tanker/opplevelser? Eller *beveger seg* fra et sted til et annet? (**Spatialisering av opplevelser**)

1.9. Har du noen gang følt at det er nesten helt umulig å ta avgjørelser, - dvs. de helt vanlige, dagligdagse? (**Ambivalens**) (NB: Gjelder ikke store avgjørelser, som jobb, boligkjøp osv.)

Oppleveres dette vedvarende og svært ubehagelig? Tviler du raskt (og sterkt) på avgjørelser du har tatt? Må du ofte gjøre om på slike avgjørelser?

Merk: Skilles fra generell svekkelse av initiativ (2.16.)

1.10. Har du noen gang vært *usikker* på hvilken type opplevelse du har, eller hvor virkelig den er? Er dette bare en forestilling/fantasi? (NB. Ikke hallusinasjoner!)

(Manglende evne til å skille opplevelseskategorier [modaliteter av intensjonalitet])

F.eks. at du var *usikker* på om du faktisk har gjort ett eller annet i fortiden (et reelt minne) eller om du bare innbiller deg det. Eller at det er vanskelig å *skille* mellom gode og vonde følelser? Eller at det er nesten umulig å vite hva slags følelse du egentlig har?

Merk: Ved klare hallusinasjoner er pasienten vanligvis *sikker* på at opplevelsen er et reelt sanseinntrykk, mens det vi her sikter til er en gjennomgående *usikkerhet* på inntrykkets art og realitet. Fenomenet er trolig vanlig ved schizofrenispektrumet, - et uttrykk for svekket ipseitet.

1.11. Har du noen gang opplevd at du nesten ikke har ”tankeenergi” - eller kraft til å tenke?
(Subjektiv motsats til observerbar svikt i målrettethet og utførelse)

(Forstyrrelse av tankeinitiativ eller -intensjonalitet)

Kan også *observeres* som nedsatt evne til å komme i gang og ordne helt dagligdagse aktiviteter som matlaging, påkledning osv.

1.12. Så noen spørsmål om oppmerksomhet. **(Oppmerksomhetsforstyrrelser)**

Subtype 1: Trollbinding: Har du noen gang følt at et eller annet (evt. en detalj) i omgivelsene dine *virkelig fanger* oppmerksomheten så sterkt at du er helt nødt til å stirre på det en god stund, selv om du ikke vil? (Ikke nok å bare være nysgjerrig eller interessert)

Subtype 2: Svekket evne til delt oppmerksomhet: Har du noen gang opplevd at det er veldig vanskelig å gjøre mer enn én ting om gangen? F.eks. snakke i telefonen samtidig som du noterer ned beskjeder? Eller følge med i trafikken når du er i en bil, og samtidig få med deg nyhetene på radioen?

1.13. Har du noen gang opplevd at hukommelsen har blitt *virkelig* dårlig, så du ikke greier å huske ting i mer enn noen få minutter? F.eks. at det er nærmest umulig å lese en bok eller se en film fordi du glemmer begynnelsen? **(Forstyrret korttidshukommelse)**

Merk: ”Short-term memory” er en *gjengs term* for den hukommelse det siktes til her. Det utelukker imidlertid ikke at det finnes andre (og tilkommer nye) klassifikasjoner.

1.14. Har noen gang tidsopplevelsen blitt annerledes? - at det er noe med tiden som ikke stemmer? – at tid ikke finnes? **(Forstyrret tidsopplevelse)**

Subtype 1: Endret *subjektiv* tidsopplevelse (time flow); at tiden raser for fort eller går for seint, står stille, virker oppstykket e.l.

Subtype 2: Endret *eksistensiell* tidsopplevelse (fortid/nåtid/fremtid); at livet bare er i *dette øyeblikket*, uten fortid eller fremtid, eller at tid egentlig ikke finnes. (Skåres ikke ved sterke følelsesmessige opplevelser.)

1.15. Har du noen gang opplevd et tomrom eller avbrudd i bevisstheten for hva du har gjort i et bestemt tidsrom? At du f.eks. plutselig er et bestemt sted uten å huske hvordan du har kommet dit. Eller er redd du i et glemt tidsrom har gjort noe ubehagelig eller ulovlig.

(Diskontinuerlig bevissthet for egne handlinger)

Overlapper dissosiativ fugue. *Merk:* Gjelder ikke det å ”falle i staver” eller ”koble ut”.

1.17. (NB. 1.17. undersøkes for 1.16. av *didaktiske grunner*) Har du noen gang opplevd at *selve språket* ditt ikke lenger fungerte som før, at du nesten ikke kunne finne de riktige ordene? Eller at du ikke kunne snakke med vanlig flyt, tempo, nøyaktighet? (Fenomenet kan evt. *observeres* som vagt eller klisjéaktig språk, - eller taushet) **(Forstyrret ekspressivt språk)**

1.16. Har du opplevd at noe du vil formidle (tema, følelse) liksom ”kom ut” helt annerledes enn du mente? Ukontrollert? Fordreid? (ikke bare ord, også mimikk, kroppsspråk) Har det hent at ansiktsuttrykket ditt forandret seg selv om du ikke ønsket det?

(Manglende samsvar mellom intendert uttrykk og det uttrykte)

Opplysninger vedrørende Domene 2: Selvbevissthet og primær tilstedeværelse

Så har jeg noen spørsmål om identitetsfølelsen og hvordan du føler deg som menneske

2.1. Har du noen gang opplevd deg selv som fremmed eller uvirkelig? Ugenkjennelig? Annerledes? Som om du manglet *kjerner* - eller identitet? – en indre *tombe*? – som om du nesten ikke eksisterer?

(Svekket følelse av ”basic self”)

(2.1 er *noe mindre spesifikt* for selvforstyrrelser enn 2.2.)

Hvis ja: har det vært slik hele livet, eller har det kommet som en forandring?

Subtype 1: start i barndom (før ca 12 år)

Subtype 2: start i ungdomstid (etter ca 12 år). Det er mulig å skåre begge subtyper.

2.2. Har du noen gang følt at det du opplever (ser, hører osv.) ikke helt er dine opplevelser? - virker fremmed eller uvirkelig (sub 1)? Dette kan øke i styrke slik at det føles som en slags avstand mellom deg selv og det du opplever (sub 2)? Eller at opplevelsen blir nesten løsrevet fra deg selv? Som om din kjerne fysisk og konkret befinner seg et sted for seg selv (sub 3)?

(Deformert 1.person-perspektiv) (Er oftest *mer spesifikt* [for selvforstyrrelser] enn 2.1.)

Subtype 1: Svekket "mineness". Pasienten opplever å ha mistet eierskapet (mineness) til tanker, følelser, sanseinntrykk og handlinger, slik at disse prosessene virker upersonlige, anonyme, livløse eller på en måte mekaniske. De har mao. mistet noe av tilknytningen til selvet. Han kan føle seg som et objekt, ikke som et menneske med sjel.

Subtype 2: Fenomenologisk distanse oppstår mellom opplevelsen og selvet, som øker slik at pasienten føler at han nærmest observerer sine tanker, sanseprosesser osv. utenfra (introspektiv selv-monitorering).

Subtype 3: Spatialisering av selvet. Selvet oppleves i disse tilfellene å ha romlig utstrekning og lokalisasjon, og på et vis være helt atskilt fra selve opplevelsen. En objektivisering, maksimal fremmedgjøring av selvet.

Følgende er en restkategori og dekker typer av depersonalisering som ikke kan skåres andre steder i domene 2:

2.3. Har du følt deg fremmed i forhold til deg selv (din psyke, atferd) på noen annen måte? (**annen depersonalisering [selv-fremmedgjøring]**). Har du noen gang vært dypt og alvorlig depriment? Hvis ja: Var depresjonen så sterk at den triste følelsen nesten var uvirkelig - ikke angikk deg?

Subtype 1: melankoliform depersonalisering forekommer ved *melankoliform depresjon* og er derfor *state*, ikke *trait*. Det er ingen forstyrrelse av "basic self" eller førsteperson-perspektivet (2.1 - *trait*).

Den depressive følelse og personen oppleves på et vis som atskilte (dissosiert), ego betrakter sin egen depresjon. Mens ved vanlig (non-melankolsk) depresjon og sorg er de to ett, og subjektet opplever *seg selv* som depriment.

Subtype 2: depersonalisering som pas. ikke kan spesifisere nærmere.

2.4. Har du noen gang følt det som om du ikke helt hører til eller er fullstendig til stede? - ikke deltar skikkelig i verden? At det er en slags avstand mellom deg og verden omkring? At folk og hendelser ikke angår deg? (Opplever i hovedsak å finne sted i *vedkommende selv*, og ikke i omgivelsene som ved derealisering. Må ikke forveksles med å "falle i staver", "falle ut" e.l.)

(Nedsatt følelse av tilstedeværelse)

Subtype 1: Kan spesifiseres: En sterk følelse av manglende tilstedeværelse i verden, med svekket naturlig gjenklang og engasjement. Inkluderer bl.a. sosial an-/hypohedoni/apati; dvs. en opplevelse av å ikke ha følelser: NB! Skåres ikke hvis sosial angst, 2.13.4.).

Subtype 2: Glassklokke-følelse. *Kan ikke spesifiseres nærmere:* En sterk fornemmelse av en usynlig vegg, og avstand til verden omkring. Som å være i en glassklokke eller bak et glass (quasi-perseptuelt).

Subtype 3: Subtype 1 eller 2 *pluss derealisering* (dvs. perseptuell forandring): Denne varianten omfatter altså både barriere-følelsen, og i tillegg konkret sanseforandring, som f.eks. blekere farger, fjernere objekter.

2.5. Har du noen gang opplevd at omgivelsene eller verden på en måte virker fremmed? Fordreid? Uvirkelig? Kunstig? Nesten som om du ser alt på film? (**Deralisering**)

(Endringene oppleves i hovedsak å finne sted i omgivelsene, ikke i vedkommende selv. Det er *levd* virkelighetsopplevelse som svekkes, ikke den begrepsbaserte realitet eller realitetstesting)

Subtype 1: Flytende, global derealisering. Vanskelig å beskrive nærmere, men verden oppleves uvirkelig, drømmeaktig, uklar, mekanisk, livløs, meningsløs.

Subtype 2: Påtrengende derealisering (hyperrealisering). Hele eller deler av verden (objekter, situasjoner) fremstår nærmest som over-virkelig, med en ubestemmelig forsterket betydning og fremtoning. Omgivelsene har en sterkt emosjonell effekt, kan f.eks. virke storslått, imponerende og gripende. (Trollbinding [1.12.1.] ofte samtidig.)

2.6. Har du noen gang følt et sterkt behov for å gruble og reflektere intenst over *deg selv* eller tankene dine? – følelsene? - handlingene? – evt. noe i omgivelsene? (svekket common sense?)

(Hyperrefleksivitet / økt refleksivitet) Overlapper med sekundær ruminasjon [1.6.2.], og deformert 1. person-perspektiv med fenomenologisk distanse [2.2.2.]

At du kanskje ikke lenger opplevde den samme letthet (lettvinthet) i livet? At det var vanskelig å være stort sett spontan og bekymringsløs?

2.7. Har du noen gang følt at det er som om du ikke er en samlet helhet, men nesten er oppsplittet/oppdelt i ulike deler? **(Jeg-spaltning)** (Denne opplevelsen kan forekomme i alle grader; se subtyper. NB: ikke tilstrekkelig å oppleve at personligheten er mangesidig.)

Subtype 1: Intervjueren (og pasienten) har en *vag uklar mistanke* om at en jeg-spaltning kan ligge bak de plagene pasienten har.

Subtype 2: "Som-om"-kvalitet. *"Plutselig kan jeg føle det som om jeg er to."*

Subtype 3: En objektivisert, romlig (spatialisert) opplevelse av jeget, men ikke psykotisk overbevisning. *"En del av meg føles fremmed og på en måte atskilt fra det normale jeg."* **Subtype 4:** Psykotisk kvalitet. *"Jeg har sluttet å spise for å sulte den fremmede delen av meg til døde."*

2.8. Har du noen gang opplevd det *som om* du observerer deg selv (eller andre) fra utsiden av din egen kropp? At du nesten er en dobbeltgjenger av deg selv? (ut-av-kroppen opplevelse)

(Dissosiativ depersonalisering)

Subtype 1: "Som-om"-kvalitet, en indre forestilling, ikke en faktisk sanseopplevelse.

Subtype 2: Dissosiativ synshallusinasjon: Pasienten kan bokstavlig talt se seg selv fra utsiden, - ikke bare som en indre forestilling?

2.9. Har du noen gang følt det som om du egentlig er en annen person enn deg selv?

(Identitetsforvirring) (Dette vil ofte være kombinert med 2.1. *Svekket følelse av "basic self"*, 2.2.

Deformert 1.person-perspektiv, samt domene 4 Transitivity; da skåres alle relevante items)

At du i korte øyeblikk kan føle det som om du er en person som du akkurat tenker på, den hunden du akkurat ser o.l.?

2.10. Har du noen gang følt det *som om* alderen din ikke stemmer? *Som om* du har en helt annen alder enn det som fremgår av fødselsattesten din? Har den følelsen vært forvirrende? Ubehaglig? (Må ikke åpenbart skyldtes de sosiale relasjoner?)

(Endring mht. opplevd kronologisk alder).

"Under en samtale følte jeg meg faktisk som en 5 år gammel liten pike."

2.11. Endret opplevelse av eget kjønn.

Subtype 2: Har du noen gang følt det som om du er av motsatt kjønn? Eller har du vært virkelig grunnleggende forvirret mht. kjønn?

Subtype 1: Føler du deg tiltrukket av eget eller motsatt kjønn? Det skal skåres kun for opplevd *frykt* for å være homofil, ikke homofili i seg selv.

2.12. Har du noen gang opplevd at du ikke lenger forstår helt selvfølgelige og naturlige ting i tilværelsen? At det er vanskelig å forstå situasjoner, mennesker, gjenstander?

(Svekket "common sense" eller "naturlig selvfølgelighet" / perpleksitet) (NB: Skåres ikke hvis hovedopplevelsen er av paranoid karakter)

Svikten kan gjelde alle domener: fysiske forhold, sosiale spilleregler, ords betydning osv. og leder ofte til hyperrefleksivitet (evt fascinasjon, nysgjerrighet).

Morbid rasjonalisme eller geometrisme er inkludert, men kreves ikke (se manualen s. 249)

2.13. Så noen spørsmål om angst/ indre uro/ stress/ ubehag. **(Angst)** (Skåres kun med +/-)

Subtype 1: Panikkanfall. Har du (noen gang hatt) sterke *anfall* av angst, helt uventet, som varer i noen minutter eller noen timer? Har du da merket skjelving, pustevansker, hjertebank, svimmelhet eller frykt for å dø, kveles, bli gal?

Subtype 2: Rent psykisk angstanfall. Hva med angstanfall *uten* noen av de nevnte kroppslige (autonome) symptomene?

Subtype 3: Fobisk angst. Har du hatt angst som blir utløst av helt spesielle ting, som åpne plasser, høyder, trange rom, spesielle dyr o.l. Hva skjer i slike situasjoner?

Subtype 4: Sosial angst. Har du vært tydelig usikker og utrygg i sosiale situasjoner eller møter, hvis andre ser på deg, hvis du har fysisk kontakt e.l.? (kan inkludere selvhenføring)

Subtype 5: Frittflytende angst. Har du hatt en mer jevn og *konstant*, diffus, frittflytende angst, spenning eller ubehag (knyttet til spesielle situasjoner eller ikke) som virkelig gjør livet vanskelig?

Subtype 6: Paranoid angst. Angst m/ paranoide forestillinger: Føler du at andre mennesker ikke vil deg godt, men vil utnytte deg, plage deg eller manipulere deg?

Neste symptom - ontologisk angst - skåres svært konservativt, - etter grundig interviu. Alminnelig angst 2.13 (se over), subtype 1, 2 eller 5 bør være tilstede samtidig. Det er derfor viktig å ha gjennomgått disse først. Likeledes bør ett av følgende være tilstede samtidig: 2.1; 2.2; 2.3; 2.5; 2.6; 2.12.

2.14. Har du noen gang følt deg *virkelig grunnleggende* engstelig, usikker og utrygg i verden, som om noe skremmende eller katastrofalt var i ferd med å skje? At menneskene og tilværelsen ikke lenger er stabile og trygge? - men snarere merkelige, uforståelige, nesten truende? (svak, underlegen, ubeslutsom?) (**Ontologisk angst**)

Merk: Vedkommende vil være mer preget av en defensiv, selvbeskyttende livsstil enn selvrealisering, og han vil nesten alltid ha en dypt forstyrret identitet.

2.15. Har du noen gang følt det som om det var et *indre slør* eller en slags blokkering i bevisstheten din? At du liksom ikke er skikkelig klar og kvikk og oppmerksom?

(Svekket klarhet [gjennomsiktighet] i bevisstheten)

Merk: Plagene skal ikke forårsakes av samtidig tankepress (1.3), hallusinasjoner, psykisk utmattelse, klinisk depresjon, organisk hjernelidelse eller stoffbruk.

Typisk utsagn: Jeg er bare i 60-70 % kontakt med verden, som om det ikke er noen skikkelig åpning ut til verden.

2.16. Har du opplevd at det er unormalt vanskelig å *komme i gang* med selv bagatellmessige ting? - at det krever en helt uvanlig kraftanstrengelse? (**Svekkelse av initiativ**)

Merk: Ren passivitet er ikke tilstrekkelig, det er *initiativet* som må være svekket. Skal heller ikke skåres hvis problemet kan forklares av mer primære vanskeligheter, som tankepress, grubling, klinisk depresjon, organiske eller farmakologiske effekter.

2.17. Har du noen gang følt at *selve evnen* til å glede seg (skåres ikke ved depresjon) har blitt borte eller svekket? Det å kunne oppleve glede når det skjer noe fint i din umiddelbare nærhet? (**Hypohedoni**)

Merk: Skilles fra sosial anhedoni (inkludert i 2.4.1) som er en *spesifisert* variant av svekket tilstedeværelse. Hypohedoni vil ofte overlappe med f.eks. svekket vitalitet (2.18), svekket tilstedeværelse (2.4) og forstyrret 1. person perspektiv (2.2). Skåre alle relevante items.

2.18. Har du noen gang hatt en følelse av å ikke være helt levende? Eller rett og slett følt deg død? En sterk og uforklarlig psykisk eller fysisk tretthet? At den indre handlekraften og spontaniteten er veldig svak eller borte? (**Svekket vitalitet**)

Subtype 1: "State"-lignende variant, inntreffer særlig i forbindelse med episoder av forverring av en ellers stasjonær tilstand/lidelse.

Subtype 2: "Trait"-lignende variant: Kan forekomme som et ganske isolert fenomen, i perioder eller mer stabilt.

Merk: Fenomenet skal ikke skåres dersom det er uttrykk for mer primære og vidtfavnende forstyrrelser som *tankepress* (1.3), *ruminasjon*, *hyperrefleksjon* (1.6), klinisk depresjon, organisk hjerneskade/sykdom eller medikamentbivirkninger.

Opplysninger vedrørende Domene 3: Kroppslige opplevelser/kroppsbewisshet

Så noen spørsmål som gjelder opplevelser knyttet til kropp og bevegelse

(Forklar kort at dette dreier seg om ganske *spesielle* og *markerte* kroppsopplevelser. Det er ellers lett å feiltolke spørsmålene til at de handler om ganske alminnelige opplevelser.)

3.1. Har du hatt episoder der du har fornemmet (eller ”sett”) at kroppen din, eller deler av den, blir mindre eller skrumper inn? Eller blir tynnere – kortere - trekker seg sammen - eller større?

(Morfologiske forandringer; endret form eller størrelse)

Subtype 1: Pasienten har en som-om-følelse av forandring, *uten* perseptuell forandring.

Subtype 2: Pasienten har en *faktisk* sansing av forandring i kroppen, han ”ser” f.eks. at hendene er forstørret (illusjon).

3.2. Har du (hatt) en sterk trang til å studere ansiktet eller kroppen din *ofte og intenst* i speilet? *Hvis ja;* var det da for å *lete etter* forandringer i ansiktet ditt, eller *så* du faktisk forandringer? Eller var det for å forsikre deg om at du eksisterer? Har du blitt overrasket eller redd for det du har sett? Eller har du tvert om prøvd å unngå ditt eget speilbilde? **(Speilrelaterte fenomen)**

Subtype 1: Pasienten leter etter forandringer (evt. uten spesielle grunn).

Subtype 2: Pasienten ser (sanseillusjon) at ansiktet er forandret eller deformert.

Subtype 3: Andre lignende fenomener, f.eks. å forsikre seg om egen eksistens.

3.3. Har du noen gang følt at kroppen din (eller deler av den) virker rar og fremmed? Livløs eller isolert? At en kroppsdel kanskje føles avspaltet eller feilstilt, eller ikke eksisterer lenger?

(Somatisk depersonalisasjon; - fremmedopplevelse av egen kropp).

3.4. Hvis du ser bort fra dette med utseende, - har du da noen gang *følt* at kroppen på en ubehagelig måte ikke passer til psyken din, eller til deg? At kropp og sjel nesten føles atskilt? **(Psyke og kropp passer ikke sammen - eller føles atskilt)**

Merk: Skåres altså ikke ved *misnøye* med kropp eller utseende i seg selv (vekt, høyde o.l.)

3.5. Enkelte mennesker kan føle at kroppen faller fra hverandre eller går i oppløsning, blir oppdelt eller forsvinner. Har du noen gang opplevd noe lignende? **(Kroppslig desintegrasjon)**

3.6. Noen mennesker forteller at de kan *merke direkte (fysiske)* de indre organene eller kroppsprosessene sine (som normalt er tause og utilgjengelig for direkte opplevelse). Har du noen gang opplevd noe slikt? (gjelder ikke alminnelige opplevelser som kraftig puls/hjertebank, tarmbevegelser eller vanlige smerter) **(Objektivisering av kroppslig opplevelse)**

Pasienten vil ellers ofte ha en tendens til å vektlegge kroppens og kroppsfunksjonenes fysiske/objektive aspekt, på bekostning av dens abstrakte, levde, ikke-romlige sider.

3.7. Mennesker har også fortalt om mange andre virkelig *rare, helt fremmede* fornemmelser og følelser i kroppen? Har du noen gang opplevd slike ting? **(Coenestasier – fordreid følesans)**

NB! Nedenfor er en veiledende liste for hva som kan forekomme, men det er ikke nødvendig å spørre etter hvert enkelt fenomen hvis pasienten avkrefter hovedspørsmålet. For fenomener som bekreftes må det spørres om en beskrivelse, hyppighet og varighet.

1. En ganske tydelig lodden/nummen/stiv følelse i armer, bein eller andre deler av kroppen?
2. En *totalt annerledes* og ny type smerte enn du har opplevd ellers? (i en spesiell del av kroppen?)
3. En eller annen fornemmelse som *flytter seg* rundt i hele kroppen?
4. En slags strøm-følelse eller elektrisk følelse?
5. En uvanlig varmfølelse eller kuldefølelse?
6. En følelse av at det er noe uvanlig som beveger seg inni kroppen?
7. En følelse av at kroppen, eller deler av kroppen, er unormalt tung eller lett eller tom, eller at du på en måte synker (tyngde) eller stiger eller svever (levitasjon)?
8. En rar og uvanlig type svimmelhet? (Vestibulært)
9. Smerte eller ubehag (overfølsom) som utløses av lyd eller forsiktig berøring (dysestesi)?

10. *Anfall* med forstyrret kroppsopplevelse, autonome vegetative forstyrrelser og dødsangst. (dysestetisk krise)

3.8. Så har jeg noen spørsmål om bevegelser og kropp: **(Motoriske forstyrrelser)**

Subtype 1: Har du noen gang opplevd at det *foles som om* kroppen (eller deler av kroppen) beveger seg, selv om den faktisk er i ro? (F. eks. hodet, et bein eller en tå) **(Pseudobevegelser)**

Subtype 2: Har du noen gang opplevd at du f.eks. går til steder - ser på ting - sier noe, uten at det var det du ville? At du faktisk gjør eller sier noe ganske annet enn det du hadde tenkt? **(Motorisk interferens)**

Merk: Slike motorisk eller verbale ”avsporinger” er en del av bevegelser som vanligvis er intendert. Eksempel: ”*automatosesyndrom*”. Må ikke representere influensfenomener (FRS).

Subtype 3: Har du noen gang opplevd at du plutselig (korte episoder) ikke kan bevege deg eller snakke? *Full blokkering*? Kan være brå, korte *anfall*, mens man er ved full bevissthet. (motsats til ”*automatosesyndrom*”; jfr subtype 2). **(Motorisk [full] lammelse/blokkering)**

Subtype 4: Har du noen gang plutselig følt deg *delvis lammet*? Tydelig *svekket eller kraftløs* i armer eller bein (ikke full blokkering)? At du kanskje haltet - eller mistet ting? Dette kan komme plutselig og vedvare i kortere eller lengre tid. **(Motorisk [delvis] lammelse/blokkering)**

Subtype 5: Har du noen gang opplevd at dagligdagse gjøremål som ellers går automatisk, plutselig ikke går av seg selv men må planlegges? **(De-automatisering)**

3.9. Har du noen gang opplevd at andre folk (evt. ting) ser ut til å beveger seg *akkurat* når du selv beveger deg? Nesten som om det er en magisk kobling mellom deg og den/det andre? Kanskje du prøver å ikke bevege deg? **(Mimetisk oppl.; resonans mellom egne og andres bevegelser)**

Opplysninger vedrørende Domene 4: Demarkasjon/transitivisme

Så til noen spørsmål om nærhet og grensene overfor andre mennesker

4.1. Når du er sammen/snakker med noen andre, har du noen gang opplevd at du ikke greier å skille helt mellom deg selv og en annen person? At dere nesten går over i hverandre? Blir sammenblandet? At du f.eks. i samtaler har vært usikker på hvilke tanker og følelser som kommer fra hvem? - følt deg invadert? **(Forveksling med annen person)**

4.2. Har du noen gang vært usikker på hvem som er hvem når du ser deg i et speil? Eller ser andres speilbilde? Eller ser i et vindusglass? Foto? Maleri? **(Forveksling med eget speilbilde)**

4.3. Har du noen gang følt det som direkte *truende eller skummelt* å bli berørt av andre, eller være fysisk nær (få en klem, ha sex)? Kanskje truende for hele din *eksistens*? **(Truende kroppskontakt)**

Merk: Gjelder også nærstående. Skal ikke skyldes tydelig paranoiditet.

Subtype 1: Utløser sterk angst og ubehag.

Subtype 2: Utløser følelse av å forsvinne, bli utslettet eller slutte å eksistere.

4.4. Har du noen gang hatt en *diffus* følelse av å være veldig, veldig utsatt og forsvarsløs i verden? Som om noe truende henger over deg? - passivisert? **(Passivitesbevissthet)**

Merk: Dette er en *diffus, ikke konkretisert følelse*, og må skilles fra psykotiske fenomener som influens (opplevelsen av å være styrt er konkret) eller paranoia. Deformert 1. person perspektiv (2.2.) kan forekomme samtidig.

4.5. Har du på noen *annen måte* følt at det er dårlige grenser mellom deg og andre mennesker, mellom deg og omverdenen? Eller vært helt spesielt opptatt av dette temaet? At du er alt for gjennomskiktig/åpen/ tynnhudet/ uten barriere (også overfor sensoriske stimuli)? Eller kanskje du har et ekstra lag *fordi* grensene er så dårlige? **(Andre transitive fenomener)**

Opplysninger vedrørende Domene 5: Eksistensiell reorientering

Til slutt vil jeg spørre litt om livsfilosofi og hvordan du ser på din egen stilling i verden.

5.1. Har du noen gang følt at alminnelige, nøytrale ting som skjer rundt deg egentlig har med deg å gjøre? - er budskap direkte til deg personlig? – som om det var en forbindelse mellom deg og det som skjer i omgivelsene (selv om du samtidig vet at dette virker usannsynlig)?

(Primære selvhenhøringsfenomener)

Merk: Opplevelsen skal ikke være *klart* sekundær til en paranoid holdning, dyp depresjon, skyldfølelse eller andre mer primære årsaker.

NB! Inkluderer følelsen av at andre kan ”lese/fornemme” ens tanker.

5.2. Har du noen gang følt at du er *belt enestående* og veldig spesiell i verden? (nb: mer enestående enn alle andre) - nesten som om du var sentrum i universet? **(Følelse av sentralitet)**

5.3. Har du noen gang følt at det bare er det du kan se med dine egne øyne som egentlig finnes? - eller er ekte? - at ingenting annet eksisterer? (Som oftest en vag og flytende følelse.)

(Følelse av at opplevelsesfeltet er eneste virkelighet)

5.4. Har du noen gang følt at du har evner eller innsikt *belt utenom det vanlige*? - kreative krefter? - om skjulte virkeligheter? – om verdens egentlige tilstand? – om menneskesinnet?

(Følelse av å ha ekstraordinære evner eller innsikt)

5.5. Har du noen gang følt det som om verden ikke finnes? – ikke er virkelig eller ekte? - bare er tilsynelatende? - en illusjon/bedrag? **(Følelse av at verden ikke eksisterer)**

5.6. Har du noen (andre) idéer eller forestillinger som du selv synes er veldig spesielle eller overnaturlige? - magiske? Har andre sagt noe? **(Magisk tenkning [om metafysisk kausalitet] som er knyttet til subjektets måte å oppleve på (5.1-5.5))**

5.7. Har interessene dine forandret seg slik at du ble veldig opptatt av f.eks. religiøse eller filosofiske tema? - eksistensielle/psykologiske/overnaturlige tema?

(Eksistensiell eller intellektuell forandring)

Merk: Skal ikke skåres ved hypomani eller mani.

5.8. Har du noen gang følt deg *belt suveren* eller overlegen i forhold til alle andre mennesker? (jfr. spesielle evner eller innsikt) – og at andre mennesker virker ganske ubetydelige for deg?

(Solipsistisk grandiositet)

Holdningen kan også av og til observeres i tale og atferd som lett manneristisk; påfallende, affektert eller teatralisk.

9. PAPERS I-IV

